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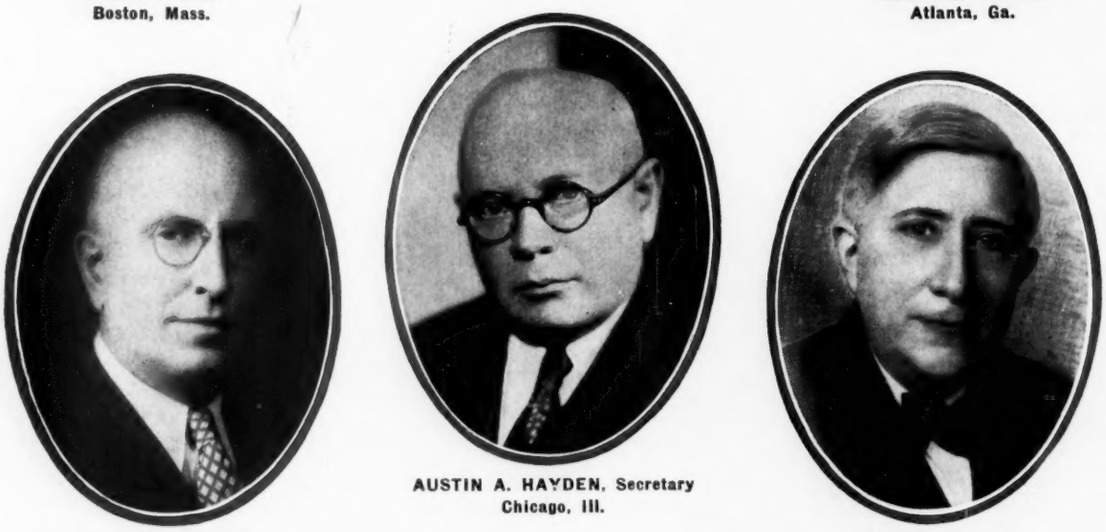

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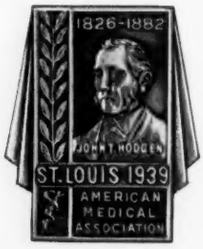

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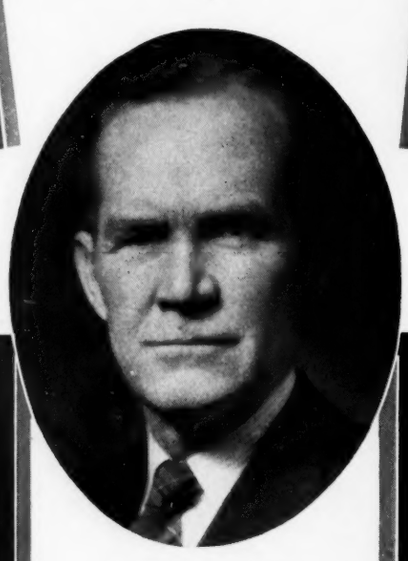

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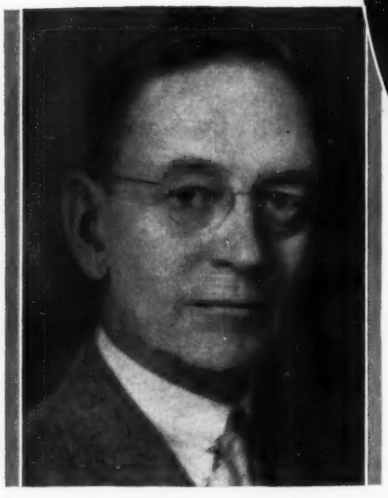

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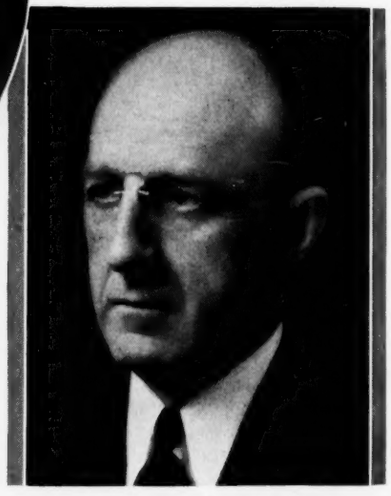

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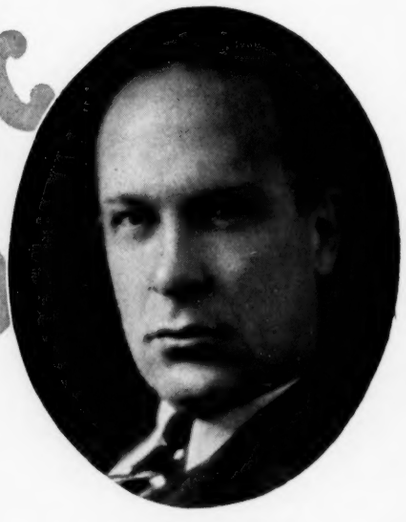

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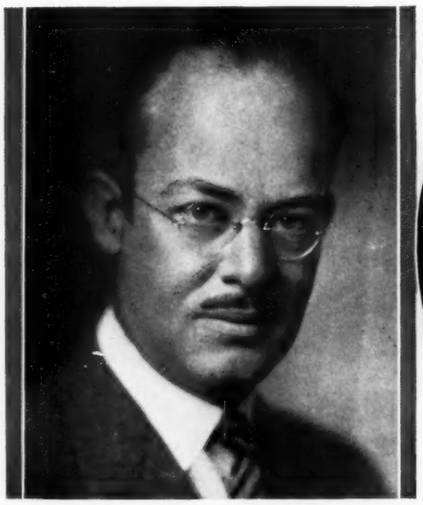

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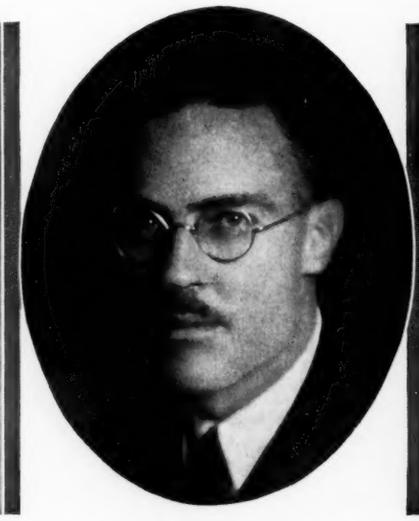

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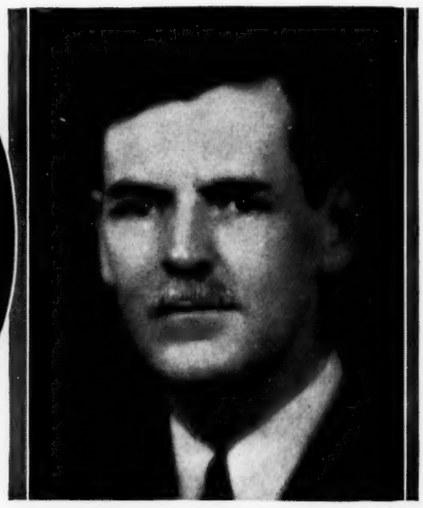

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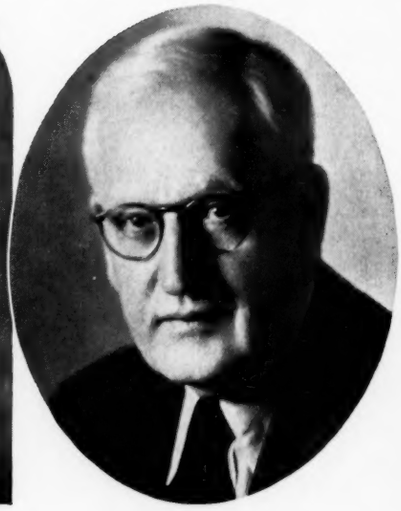

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ANGINA PECTORIS AND CARDIAC INFARCTION FROM TRAUMA OR UNUSUAL EFFORT

WITH A CONSIDERATION OF CERTAIN MEDICOLEGAL ASPECTS

ERNST P. BOAS, M.D.

NEW YORK

Can a nonpenetrating injury to the chest or severe bodily effort directly provoke cardiac infarction or lead to changes in the heart that give rise to the syndrome of angina pectoris? This question, arising so frequently in connection with claims under workmen's compensation acts, has not yet been satisfactorily answered. The preponderant opinion at present seems to be that trauma and effort so rarely precede the onset of coronary thrombosis that their association is just coincidental. This thesis has been particularly elaborated by Master and his associates.[1] They have analyzed the events attending 530 attacks of coronary thrombosis. They find that 22 per cent of attacks occur when the patient is at rest, 20 per cent while he is asleep, 18 per cent while he is walking, 14 per cent during mild activity and 5 per cent during moderate activity. They point out that since half of the day of normal persons is spent in mild or moderate activity, one might well expect that half of all attacks of coronary thrombosis would occur during this period. The fact that 36.9 per cent of attacks occurred during such mild activity therefore is interpreted as a mere temporal, not a causal, association. Only 2 per cent of the attacks followed unusual exertion, and 5 per cent followed excitement. On the basis of this low incidence of coronary thrombosis following unusual stress the authors conclude that in such cases the association of the stress and the vascular occlusion is only accidental. Coronary thrombosis following operation was encountered in 4 per cent of their series. In this instance, however, the authors do not apply the criteria of probability employed in their discussion of the effect of exertion on coronary thrombosis but on general grounds conclude that operation is a factor that may initiate coronary thrombosis.[2]

These deductions are inconsistent. I fully agree with Master that coronary thrombosis very often occurs while the individual is at rest or asleep and that in many cases no antecedent injury or effort has taken place. But to argue from this that such accidents are not competent causes of coronary thrombosis appears incorrect. The fact that pneumothorax is due usually to pulmonary tuberculosis or to the rupture of an emphysematous bleb does not negate the fact that it may follow trauma to the chest; or that cerebral hemorrhage is caused usually by disease of the cerebral arteries that it may not be due to trauma to the head.

In my experience trauma or bodily effort may directly induce closure of a coronary artery with cardiac infarction or may initiate the syndrome of angina pectoris. This occurs with sufficient frequency to be of practical clinical importance as well as of medicolegal significance. I propose in this paper to present evidence from the literature as well as from my personal experience that injuries to the chest or severe effort may produce signs and symptoms of injury or impairment of the coronary arterial circulation.

TRAUMATIC NONPENETRATING INJURIES OF THE HEART

Many cases of injury to the myocardium following nonpenetrating injuries to the chest have been observed at necropsy and reported in the literature. In Warburg's [3] recent monograph 174 instances of myocardial lesions due to nonpenetrating traumatic injuries to the chest are abstracted from the literature. To these he adds thirteen new cases of his own. Many types of myocardial injury are described, ranging from rupture of one of the cardiac chambers to scattered small myocardial hemorrhages. In a number of the cases in his series, as well as in others not included in his book, damage to the coronary arteries or the anginal syndrome has been observed after this type of injury. A few such cases will be briefly cited.

A man aged 45, struck in the precordium by a wagon shaft, was unconscious for several minutes and when consciousness returned complained of severe pain in the chest, back, left shoulder and left arm.[4] Physical examination revealed no cardiac abnormalities. The patient continued to suffer from cardiac pain, which came particularly when he was active and which prevented him from climbing stairs. He died suddenly six months after the accident. At necropsy organized thrombi were found in the anterior descending branch of the left and in the circumflex branch of the right coronary artery. The myocardium was scarred and there was an aneurysm of the left ventricle at the apex. There was advanced arteriosclerosis of the descending aorta between the aortic isthmus and the diaphragm.

A boy aged 12 years was run over by a wagon and six ribs of the left side of the chest were fractured.[5] After recovery from the accident the boy was apparently well for twelve years, except

1. Master, A. M.; Dack, Simon, and Jaffe, H. L.: Factors and Events Associated with Onset of Coronary Artery Thrombosis, J. A. M. A. **109:** 536 (Aug. 21) 1937.

2. Master, A. M.; Dack, Simon, and Jaffe, H. L.: Postoperative Coronary Occlusion, J. A. M. A. **110:** 1415 (April 30) 1938.

3. Warburg, Erik: Subacute and Chronic Pericardial and Myocardial Lesions Due to Non-Penetrating Traumatic Injuries, New York, Oxford University Press, 1938.

4. Schmincke, A.: Beitr. z. traumatischen Aetiologie d. Arteriosclerose, Deutsches Arch. f. klin. Med. **149:** 145 (Dec.) 1925.

5. Joachim, H., and Mays, A. T.: A Case of Cardiac Aneurysm Probably of Traumatic Origin, Am. Heart J. **2:** 682 (Aug.) 1927.

for occasional attacks of palpitation and vomiting. At the age of 24 he began having attacks of paroxysmal auricular flutter, some of which lasted as long as a week. Heart failure developed in some of the attacks. He died suddenly at the age of 25. Necropsy revealed an aneurysm of the anterior aspect of the left ventricle. The anterior descending branch of the left coronary artery stopped abruptly at the upper edge of the aneurysm. The authors concluded that it had been destroyed by the trauma thirteen years previously.

A man aged 40 who had been perfectly well fell on his back while skiing.[6] When he arose he felt nauseated, but he kept on. A quarter of an hour later he collapsed, vomited and complained of severe precordial pressure and angor animi. The pulse was irregular, the heart sounds faint and the blood pressure 95 systolic, 55 diastolic. In a short time the liver became enlarged. Fever and leukocytosis followed. An electrocardiogram taken twenty-four hours after the fall showed a high take-off of the T waves in leads 2 and 3. A subsequent electrocardiogram showed a typical Q_3-T_3 pattern.

A man aged 57, who was accidentally struck over the precordium by a golf ball, was in a state of shock for fifteen minutes but gradually recovered, so that he was able to attend to his business the following two days.[7] On the night of the third day there was an attack of angina pectoris, with electrocardiographic evidence of cardiac infarction. Prior to the injury there had been no symptoms of coronary disease.

Bean[8] described three cases of coronary thrombosis following trauma. Beck[9] as well as Munck[10] has described contusions of the heart with hemorrhage into the myocardium, verified at necropsy.

Experimental studies have confirmed these clinical observations. Kissane, Fidler and Koons[11] struck blows on the chests of anesthetized dogs. Electrocardiographic abnormalities, especially changes in the RT segments, were observed. Pathologic studies revealed subendocardial and subpericardial hemorrhages. In three cases in which the electrocardiograms were abnormal, no lesions were discovered. The most complete studies are those of Schlomka.[12] Most of the experiments were carried out on rabbits and cats; in two cases dogs were used. The precordium was struck with blows of moderate intensity. Rarely was the blow sufficiently strong to cause rupture of the heart or death. Frequent electrocardiograms were taken. Characteristic and common observations were sinus bradycardia (even when the vagus had previously been cut), extrasystoles, ventricular tachycardia, ventricular fibrillation, auricular fibrillation and partial and complete heart block. In many instances typical "coronary" T waves, with high take-offs, were encountered. X-ray studies usually revealed cardiac dilatation. In many cases the electrocardiographic changes rapidly returned to normal; in not a few there was permanent cardiac damage, manifested by persistent electrocardiographic changes and cardiac enlargement. Cardiac abnormalities occurred particularly when the precordium itself was struck; injuries to other parts of the chest rarely produced cardiac damage. T wave changes occurred with particular frequency from a quick sharp blow with a small object concentrated in a small area, particularly directly over the sternum or the apex. Pathologic changes were chiefly focal myocardial hemorrhages. In some animals who succumbed to the trauma no anatomic lesions were discovered. Some of the rabbits used had previously been sensitized to horse serum. In these, anemic infarcts of the myocardium were very common. From his own studies and from a review of the clinical, physiologic and pathologic literature Schlomka concluded that blows sharply localized to the precordial area produce a spasm of the coronary arteries and that, depending on the duration of the ischemia, the changes in the myocardium may be reversible or irreversible. The coronary arteries themselves may be injured by the ischemia so that permanent damage to these vessels may ensue. He drew a parallel between such coronary spasms and the segmental arterial spasm observed in the case of gunshot wounds that penetrate the tissues near arteries of the extremities. Arterial spasm following arterial embolism may offer an analogous phenomenon.

Kohn[13] was the first to describe the syndrome of traumatic angina pectoris. Careful students of the subject such as Warburg,[3] Schlomka,[12] Beck[9] and Barber[14] all agree that symptoms of angina pectoris may be initiated by traumatic nonpenetrating injuries to the chest in the precordial region. I entertain the same conviction. The following case, which I observed, offers convincing evidence of the validity of this conclusion:

CASE 1.—A man aged 62, who had worked for seventeen years as a plumber, doing heavy work, carrying bathtubs into buildings and the like, without any symptoms whatever, was cleaning a sewer pipe, Nov. 7, 1935, which had become blocked. For this work a heavy cable was fed into the sewer by one man and extracted on the other side by the patient. The cable was new and had considerable spring. The patient was winding the cable into a coil on the receiving end when it suddenly sprang and struck him with considerable force in the left lower part of the chest. He was stunned. Things became dark before his eyes and he had to remain leaning against the wall for half an hour. Then with some effort he finished the job but went to the feeding end of the cable, where the work is lighter. He managed to work slowly and intermittently for the rest of the day. In the afternoon local pain and swelling appeared on his chest where he was struck, and when he returned home his wife remarked that his face looked pale and drawn. The next morning he returned to work, but slight effort, such as using a wrench, provoked pain in the precordium, compelling him to rest every half hour or so. On this day and on the following days he noted increasing difficulty in walking, so that by the fourth day he was compelled to stop, after walking five blocks, by a sense of choking in the left side of the chest, which radiated to the left side of the neck. He continued to work with difficulty for ten days. November 25 he experienced precordial pain which become progressively more severe, which lasted all night and for which he was given a hypodermic injection of morphine. He remained in bed eight weeks after this attack. Ever since he has had angina pectoris on effort. Fluoroscopic examination June 24, 1938, revealed much enlargement of the left ventricle. The first heart sound was of good quality. There was a systolic murmur at the apex. The blood pressure was 110 systolic, 80 diastolic. The electrocardiogram showed low voltage and slurring of the QRS complex in all leads, a Q wave and a flat T wave in lead 1, and a large Q wave and negative T wave in lead 4.

6. Kienle, F.: Klinische und elektrokardiographische Beobachtungen bei traumatischem Hinterwandinfarkt, Ztschr. f. Kreislaufforsch. **30:** 674 (Sept. 15) 1938.

7. Relation of Myocarditis and Coronary Thrombosis to Trauma, Queries and Minor Notes, J. A. M. A. **101:** 1503 (Nov. 4) 1933.

8. Bean, W. B.: Infarction of the Heart, a Morphological and Clinical Appraisal of 300 Cases: I. Predisposing and Precipitating Conditions, Am. Heart J. **14:** 684 (Dec.) 1937.

9. Bright, E. F., and Beck, C. S.: Nonpenetrating Wounds of the Heart, Am. Heart J. **10:** 293 (Feb.) 1935. Beck, C. S.: Contusions of the Heart, J. A. M. A. **104:** 109 (Jan. 12) 1935.

10. Munck, Willy: Investigations on Heart Lesions Due to Blunt Force, Hospitalstid. **80:** 525 (May 11) 1937.

11. Kissane, R. W.; Fidler, R. S., and Koons, R. A.: Electrocardiographic Changes Following External Chest Injuries to Dogs, Ann. Int. Med. **11:** 907 (Dec.) 1938.

12. Schlomka, I.: Commotio Cordis und ihre Folgen, Ergebn. d. inn. Med. u. Kinderh. **47:** 1, 1934.

13. Kohn, H.: Angina Pectoris und Unfall, Klin. Wchnschr. **8:** 795 (April 23), 843 (April 30) 1929.

14. Barber, Hugh: Trauma of the Heart, Brit. M. J. **1:** 433 (Feb. 26) 1938.

CORONARY OCCLUSION OR ANGINA PECTORIS INDUCED BY SEVERE OR UNUSUAL BODILY EFFORT

Ever since Kohn in 1929 set up the syndrome of angina pectoris traumatica there have been repeated references in the literature to coronary artery occlusion following severe bodily effort. Fitzhugh and Hamilton[15] described many cases of coronary thrombosis following unusual or violent physical exertion and concluded that the effort induced the arterial occlusion. In their experience "more often than not fatal anginas or coronary occlusions were immediately preceded by unusual departures from ordinary habits of living."

Kampmann[16] described the case of a man aged 43 who was pulled to the ground violently when the crank handle of a bus that he was cranking kicked back. Immediately he felt sharp precordial pain and became dizzy. He was able to drive a little farther but half an hour after the accident vomited and experienced severe precordial pain, broke out in a cold sweat and became pulseless and cyanotic. He slowly improved. The following day his heart rate was 32. In the subsequent eight days it rose slowly to 60, but he had a number of attacks of Stokes-Adams syncope. Two years later he still had precordial pain on exertion. The heart sounds were faint; the electrocardiogram showed a Q_3-T_3 pattern and a PR interval of 0.21 second. This man experienced an infarction of the posterior aspect of his left ventricle after the accident and with this a complete heart block. The author referred to other reported cases of traumatic heart block.

Phipps,[17] without giving detailed clinical histories that make possible a critical evaluation of his data, reported that in 40 per cent of cases coronary thrombosis occurred under conditions of physical stress and that in 13 per cent it occurred during exercise. Luten[18] found effort rarely associated with coronary thrombosis, but it is chiefly Master who denies that effort is a competent producing cause of coronary thrombosis.

Bean and Mills[19] in a recent study of 1,640 attacks of coronary occlusion found that in northern cities these attacks occur with much greater frequency in winter than in summer. There were twice as many attacks in January as in August. They conclude that the greater frequency of infections and the heightened metabolism in cold weather increase the work of the heart and so add to the hazards to which these patients are exposed.

I have examined and studied twenty-five patients in whom coronary occlusion appears to have been directly induced by severe or unusual exertion. The following cases illustrate this sequence:

CASE 2.—I. R., a man aged 47, seen July 27, 1937, had known for about a year that his blood pressure ranged between 150 and 160. He had, however, felt quite well except for headaches and had worked hard as a painter without symptoms. July 6, while lifting an 85 pound can of paint, he experienced sudden pain in the left arm and in both jaws. He dropped the can and in a few moments felt well. Five hours later, while he was rolling a barrel up an incline, he experienced some pain in the left arm and jaw, as well as in the midchest. The pain became very severe, lasted eight hours and was associated with a sense of choking. He remained in bed for three weeks, until the time of his examination. He was a well built man. Fluoroscopic examination revealed slight enlargement of the left ventricle. The heart sounds were of good quality. There were no murmurs. The blood pressure was 130 systolic, 80 diastolic. The electrocardiogram showed Q waves and sharply negative T waves in leads 2 and 3. He was again seen two months later. He had no symptoms, but the blood pressure had risen to 160 systolic and 100 diastolic. He was awarded compensation under the workmen's compensation law of New York State.

CASE 3.—M. S., a man aged 53, who had always been perfectly well and strong and had no knowledge of previous heart disease or high blood pressure, at about 9:30 a. m. May 22, 1937, lifted a roll of pattern paper, weighing 180 pounds, in order to place it on a table. He experienced sudden sharp and pressing pain in the left lower part of the chest, which radiated to the back of the left side of the chest. For a few minutes he was unable to catch his breath and unable to straighten up. Then he coughed up a small amount of blood with some relief. He immediately stopped work and was driven home by a friend. Severe pain continued throughout the day, in spite of the fact that he received a number of hypodermic injections. An electrocardiogram taken May 24 showed an elevation of the RT segment in leads 1 and 2, a marked depression of the RT segment, a negative T wave and a deep Q wave in lead 4. May 27 he was taken to the Manhattan General Hospital, where he stayed until July 12. Here a diagnosis of coronary thrombosis was made. An electrocardiogram taken July 12 showed negative cove plane T waves in leads 1 and 2, very low voltage in all leads, Q waves in leads 2 and 3 and a negative T wave and a Q wave in lead 4. August 22 he was admitted to the Israel-Zion Hospital, complaining of shortness of breath and precordial pain, and remained in this hospital until October 14. Here a diagnosis of left ventricular insufficiency was made in addition to that of coronary thrombosis. He has been in bed most of the time since then. When he attempts to walk he experiences precordial pain and pain in the back which compel him to rest. Bending too provokes such pain. Sept. 20, 1938, fluoroscopic and x-ray examination revealed marked generalized dilatation of the aorta and a great enlargement of the heart, involving chiefly the left ventricle and auricle. Along the lower half of the left border of the heart there was a marked bulge, which on fluoroscopic examination showed practically no pulsation. On the x-ray film one saw marking the edge of this bulge a crescentic shadow, which apparently represented calcification. The heart sounds were feeble. There were no murmurs. The blood pressure was 130 systolic, 90 diastolic. The electrocardiogram showed low voltage in all leads, Q waves in leads 1, 2 and 4, an elevated RT segment, a negative T wave in leads 1 and 4 and a W shaped QRS complex in leads 2 and 3.

CASE 4.—S. N., a man aged 44, first observed April 2, 1937, had had occasional attacks of midabdominal cramps three years previously unrelated to meals. At that time he was unable to walk or climb stairs as readily as formerly, because he became tired and slightly dyspneic. After a few months these symptoms disappeared, but they recurred in January 1937. A gastrointestinal x-ray series suggested the presence of a peptic ulcer. In March he noted a heaviness in his chest, which was persistent but was not intensified by excitement. March 29, while lifting a 50 pound package, he experienced severe substernal pain, was unable to catch his breath and collapsed. The pain lasted about fifteen minues. He went home by subway, stayed in bed for one day and had no further pain. He was seen four days after the accident. Fluoroscopic examination revealed a heart of normal size and configuration. The heart sounds were dull. There were no murmurs. The blood pressure was 190 systolic, 60 diastolic. The electrocardiogram showed elevation of the RT segment in leads 1, 2 and 4. An electrocardiogram taken April 16 showed a diphasic T wave in lead 1, a depressed RT segment in leads 2 and 3 and an elevated RT segment in lead 4. He was seen at intervals during the following one and a half years, and during this time no additional symptoms developed. He applied for compensation under the workmen's compensation act of New York state but his claim was disallowed.

15. Fitzhugh, Greene, and Hamilton, B. E.: Coronary Occlusion and Fatal Angina Pectoris: Study of the Immediate Causes and Their Prevention, J. A. M. A. **100:** 475 (Feb. 18) 1933.
16. Kampmann, Werner: Ein Fall von isolierter Verletzung des Herzreitzleitungssystems, München. med. Wchnschr. **82:** 129 (Jan. 24) 1935.
17. Phipps, Cadis: Contributory Causes of Coronary Thrombosis, J. A. M. A. **106:** 761 (March 7) 1936.
18. Luten, Drew: Contributory Factors in Coronary Occlusion, Am. Heart J. **7:** 36 (Oct.) 1931.
19. Bean, W. B., and Mills, C. A.: Coronary Occlusion, Heart Failure and Environmental Temperatures, Am. Heart J. **16:** 701 (Dec.) 1938.

CASE 5.—S. S., a man aged 49, first observed Nov. 18, 1935, had been perfectly well until three weeks previously, when, while carrying a 60 pound package up one flight from the cellar, he experienced substernal pressure, which lasted about five minutes. This was unassociated with sweating. He continued work but from that time on noted that on working too hard or on walking three blocks he would experience substernal pressure and pain, which would compel him to stop his activities. He was well built. Fluoroscopic examination revealed a heart of normal size and configuration. The first heart sound was split. There were no murmurs. The blood pressure was 130 systolic, 90 diastolic. The electrocardiogram was normal except for a very low T wave in lead 4. He was seen again in February 1936; the symptoms were unchanged. The T wave in lead 4 was now of normal size. He was last seen Nov. 20, 1936. His anginal symptoms persisted.

CASE 6.—H. T. in 1926, when he was 35 years old, was driving a new truck and the motor stalled. He had to crank the engine. This took some time and effort because the motor was new. When he got back into his seat and began to drive away, he experienced pain in the region of the left elbow and some distress in the left side of the chest and had difficulty in catching his breath. He was compelled to stop the truck and get out and walk to the nearest subway station. The original elbow pain stopped after about two minutes but recurred as he walked and compelled him to make his way slowly. He then rested for five weeks and felt quite well until 1936. He had changed to a lighter occupation. In 1936 he began to experience substernal pressure and difficulty in breathing on walking a few blocks, compelling him to rest. These symptoms have continued. I examined him in May 1938. Fluoroscopic examination revealed a transverse heart of normal size. The first heart sound was a bit dull. There were no murmurs. The blood pressure was 140 systolic, 90 diastolic. The electrocardiogram was normal. He was seen again in October 1938. The symptoms of angina pectoris had persisted, but there had been no prolonged attacks of pain. Physical examination disclosed no new developments, but the electrocardiogram now showed a depressed RT segment in lead 1 and a sharply negative T wave in lead 4.

CASE 7.—P. L., a man aged 43, who had worked at his occupation as a painter without any symptoms whatever until July 12, 1937, on this day started work an hour late and rushed to catch up. He was sizing some partitions with a sponge and working very fast. After forty-five minutes he began to sweat and became dizzy. He then experienced a tearing pain to the left of the sternum, which was followed by a constricting sensation, and broke out into a cold sweat. Hypodermic medication was given and he was taken to a hospital, where he stayed for four weeks and where an electrocardiogram showing infarction of the left ventricle was obtained. After discharge from the hospital he still had frequent precordial pain, unrelated to exertion, although greater exertion caused some difficulty in breathing. He was, however, able to resume his work. Fluoroscopic examination September 28 revealed slight enlargement of the left ventricle and some dilatation and elongation of the aorta. The heart sounds were of good quality. There were no murmurs. The blood pressure was 120 systolic, 70 diastolic. The electrocardiogram showed a flat T wave in lead 2 and a sharply negative T wave in lead 3. He applied for compensation under the workmen's compensation laws of New York state, but his claim was disallowed.

CASE 8.—J. B., a man aged 48, first observed Dec. 22, 1936, had had an attack of pressure in the upper part of the abdomen, sweating and collapse five weeks previously, which lasted for ten minutes. He was in bed for ten days and had no recurrence of the pain. He was then well and was working as a hotel keeper. December 20 he had an argument with two drunken men and threw them downstairs and immediately afterward dragged a drunken woman up two flights of stairs. When he reached the top of the stairs he broke out into a cold sweat and was unable to move. He then experienced substernal pain and difficulty in breathing. He was seen two days after this occurrence. He was febrile and appeared ill. There were a few rales at the bases of the lungs. The heart sounds were feeble. The heart rate was rapid. He was seen subsequently on March 25, 1937. Fluoroscopic examination revealed much enlargement of the left ventricle and some enlargement of the left auricle. The first heart sound was faint. The pulmonic second sound was accentuated. There were no murmurs. The blood pressure was 90 systolic, 60 diastolic. The electrocardiogram showed low voltage and slurring of the QRS complex in all leads and Q waves and negative T waves in leads 1 and 4.

CASE 9.—B. S., a man aged 55, first seen in February 1938, was suddenly seized nine years previously, while dancing after a wedding dinner, with substernal pressure which developed into severe pain and was associated with cold sweat. The pain lasted about four hours and was relieved by hypodermic medication. He was in bed for eight weeks. Ever since, walking two blocks would provoke substernal pressure, compelling him to rest. During the three months prior to his present examination, anginal attacks would come on at rest and there had been a number of attacks of paroxysmal dyspnea. Physical examination revealed a few rales at the bases of both lungs. Fluoroscopic examination revealed great enlargement of the left ventricle and of the left auricle. The first heart sound was quite faint and split. The second sound at the apex was reduplicated. There was a systolic murmur at the apex. The blood pressure was 115 systolic, 70 diastolic. The electrocardiogram revealed bundle branch block.

CASE 10.—M. E., a man aged 47, first observed in December 1934, for several years had had some heartburn after eating but was otherwise well. Eleven weeks previously, during intercourse, he experienced pressing pain to the left of the sternum, which radiated down the left arm, and he broke out into a cold sweat. The pain lasted about fifteen minutes and was relieved by a hypodermic injection. He went to work the next day; he again broke out into a cold sweat, feared that another attack was coming on and went to a hospital, where he stayed for four weeks. After discharge from the hospital he noted that walking six blocks or so during cold weather would provoke substernal pressure which compelled him to stop. Fluoroscopic examination revealed some enlargement of the left ventricle. The first heart sound was dull. Both basic second sounds were accentuated. There was a systolic murmur at the aortic area. The blood pressure was 110 systolic, 80 diastolic. The electrocardiogram showed an elevated RT segment with a diphasic T wave and a Q wave in lead 1, a slightly depressed RT segment with a diphasic T wave in lead 3 and a deep Q wave and a negative T wave in lead 4. He was seen again in December 1937, still complaining of angina pectoris on exertion. The electrocardiogram now showed a sharply negative T wave in lead 1, and the T wave in lead 4 had become positive.

CASE 11.—A. N., a man aged 47, who had in July 1930 experienced slight distress in the lower anterior part of the chest while playing ball, would stop when experiencing pain. Oct. 18, 1937, while playing football, he experienced sudden pain in the anterior part of the chest, as though he were in a vice, and broke out into a cold sweat. Severe pain lasted one-half hour and milder pain lasted for two days. The following day his temperature rose to 101 F. He stayed in bed for six days. After this he noted that walking or climbing stairs would provoke substernal pain, compelling him to rest. He was a stockily built man. Fluoroscopic examination October 28 revealed moderate enlargement of the left ventricle and left auricle. The first heart sound was dull. There were no murmurs. The blood pressure was 110 systolic, 70 diastolic. The electrocardiogram showed slurring of the QRS complex and Q waves and sharply negative T waves in leads 2 and 3.

CASE 12.—I. D., a man aged 49, who had been complaining for one and a half years of epigastric burning one or two hours after meals, relieved by sodium bicarbonate or milk, occasionally was awakened at night by this pain. Jan. 18, 1936, while trudging through a heavy snowfall on a very cold day, he began to experience cramplike pain behind the sternum, which radiated to the left upper extremity. He began to sweat. The pain slowly became worse, and he returned home and was given a hypodermic injection a few hours later. Intermittent pain continued for two days and he remained in bed for two weeks. He was seen February 8. He was a stout man of high color. Fluoroscopic examination revealed a heart of normal size and configuration. The heart sounds were of good quality. There were no murmurs. The blood pressure was 135 systolic, 80 diastolic. The

electrocardiogram showed a small Q wave and a low T wave in lead 1 and a deep Q wave and sharply negative T wave in lead 4.

CASE 13.—M. B., a man aged 45, had had a very hard day Sept. 21, 1938, delivering furniture. It was raining heavily and he was wet to the skin. Indeed the weather was so inclement that the driver of the furniture truck telephoned his employer a number of times asking for permission to postpone deliveries to another day, but permission was refused. Toward the end of the day, while the patient was carrying a chifforobe up a second flight of stairs and while he was lifting it high over the banister to make a turn at the landing, he suddenly felt as though something had snapped behind the lower part of the sternum. This was immediately followed by severe pressure across the lower part of the chest, which radiated to the lower pectoral area. He made his way down the stairs, collapsed and was unconscious for four hours. He was taken to a hospital, where he remained for six weeks. After his discharge from the hospital, walking five blocks always provoked sharp parasternal pain on the left, which radiated to the back and which compelled him to rest. Fluoroscopic examination December 30 revealed slight enlargement of both ventricles. The heart sounds were dull. There were no murmurs. The blood pressure was 95 systolic, 70 diastolic. The electrocardiogram had a very low T wave in lead 1 and a Q wave and negative T wave in lead 4.

CASE 14.—S. S., first observed in August 1930, when he was 58, at that time had no symptoms referable to the heart. He had an old tuberculous lesion at both apexes. The left ventricle was slightly enlarged and the aorta a bit dilated and dense. The heart sounds were of good quality. The blood pressure was 160 systolic, 100 diastolic. The electrocardiogram showed only left axis deviation and low voltage. In November 1934 he began to experience substernal pressure and epigastric pain while at work and while walking. The pain would compel him to rest. Feb. 22, 1935, while walking to work during a blizzard, he experienced severe substernal pressure and great weakness. Nevertheless he went to his shop and went to work, and four hours later, while lifting a heavy machine, he became very weak, broke out into a profuse sweat and fainted. Fluoroscopic examination four days later, February 26, revealed that the heart was of the vertical type and not enlarged. The heart sounds were of good quality. There was a systolic murmur at the apex. The blood pressure was 140 systolic, 80 diastolic. The electrocardiogram showed a depressed RT segment, with a diphasic T wave in lead 2 and a Q wave and sharply negative T wave in lead 3. Angina pectoris on slight exertion persisted. He applied for compensation under the workmen's compensation laws of New York State. This case differs from the others in that the symptoms suggesting coronary thrombosis occurred four hours before he went to work and were apparently precipitated by the effort of walking in a snow storm. The final collapse occurred when he overexerted himself four hours after the beginning of the symptoms of coronary thrombosis. In this case the exertion of walking in the snow storm rather than the lifting of a heavy machine must be regarded as the cause of the coronary thrombosis.

These clinical observations, together with those of other authors, warrant the conclusion that the syndrome of coronary occlusion may be induced by a nonpenetrating injury to the chest or by unusual effort and that the same causes may initiate the syndrome of angina pectoris. The reluctance of many clinicians to accept a causal relationship between these phenomena is due in part to the fact that when an injury occurs during work the patient naturally seeks to establish that the work as such was responsible for his disability. He may wittingly or unwittingly construct a logical series of events leading up to his injury. There is a universal tendency among patients to ascribe their illnesses to some concrete indiscretion or accident. Of the fourteen cases that I have presented, only five involved a problem of workmen's compensation. In the others the patient had nothing to gain by ascribing his illness to an unusual effort.

Inability to visualize the mechanism by which lesions of the coronary arteries may be induced by trauma or by bodily effort has further retarded the acceptance of a causal relationship between these events. Recent pathologic studies throw some light on this problem. The manner in which nonpenetrating injuries of the chest can cause myocardial damage has been discussed, and experimental and postmortem evidence has been presented to prove that such traumatic lesions of the heart actually occur.

The way in which unusual bodily effort or strain can induce acute changes in the coronary arteries and in the coronary circulation is suggested by several anatomic studies. Leary [20] states that rupture of an atheromatous cavity of the wall of a coronary artery into the lumen is the standard terminal lesion in older persons dying of coronary thrombosis.

Paterson [21] has studied intimal hemorrhages of the coronary arteries, which are due apparently to rupture of capillaries in the arterial wall. Such intimal hemorrhages are commonly found in recently thrombosed coronary arteries. His studies suggest that capillary rupture and intimal hemorrhage are closely concerned with the formation of most coronary thrombi. Thrombosis in the lumen of a coronary artery may be initiated by diffusion of blood from an intimal hemorrhage into the lumen, by necrosis of the intima or by retrograde capillary thrombosis. Paterson points out that during periods of physical stress a rise in pressure in the capillaries of the intima may occur, causing their rupture. Complete closure of a coronary artery by a thrombus may occur hours or days after the original capillary hemorrhage.

Winternitz [22] has demonstrated the rich capillary network that is found in the arterial wall, particularly in the neighborhood of sclerotic areas, and has shown that hemorrhages into the intima are frequently the precipitating factors in arterial thrombosis.

Wartman [23] has described six cases of complete occlusion of a sclerotic coronary artery by a large hemorrhage into its wall. In one of these cases an intra-arterial thrombus was associated with the intramural hemorrhage. In all cases death was caused by the hemorrhagic arterial occlusion. Winternitz [22] observed similar cases.

These anatomic studies suggest how unusual exertion may bring about coronary artery occlusion. With physical effort there is a sudden alteration of arterial pressure, cardiac action is increased, rupture of one of the capillaries or sinusoids in the arterial wall may occur, or a softened atheromatous plaque may rupture into the arterial lumen. If the hemorrhage is large there may be almost immediate occlusion of a coronary artery; if the hemorrhage is small or slow or if it is followed by a gradually growing mural thrombus the occlusion may develop slowly or remain incomplete. Thus the concept of coronary occlusion and of angina pectoris induced by physical exertion established by

20. Leary, Timothy: Coronary Spasm as a Possible Factor in Producing Sudden Death, Am. Heart J. **10**: 338 (Feb.) 1935.

21. Paterson, J. C.: Capillary Rupture with Intimal Hemorrhage as a Causative Factor in Coronary Thrombosis, Arch. Path. **25**: 474 (April) 1938. Paterson, J. C.: Relation of Physical Exertion and Emotion to Precipitation of Coronary Thrombi, J. A. M. A. **112**: 895 (March 11) 1939.

22. Winternitz, M. C.; Thomas, R. M., and LeCompte, P. M.: The Biology of Arteriosclerosis, Springfield, Ill., Charles C. Thomas, Publisher, 1938.

23. Wartman, W. B.: Occlusion of Coronary Arteries by Hemorrhage into Their Walls, Am. Heart. J. **15**: 459 (April) 1938.

discriminating clinical observation finds support and confirmation in dissections of diseased arteries. It follows that when symptoms of coronary artery disease are induced by physical strain, even when the patient has had no preceding symptoms, there have most likely been antecedent anatomic lesions of the coronary arteries. When such symptoms follow nonpenetrating blows to the precordium no such preexisting arterial disease need be predicated.

CRITERIA FOR DIAGNOSIS

These cases often give rise to legal issues, in particular in connection with workmen's compensation acts. The question arises whether the particular injury or effort was a competent producing cause of the symptoms. This can be answered only by a careful evaluation of the succession of events in each individual case, with due recognition of the fact that patients tend to ascribe their illnesses to some unusual experience which actually may have no significance.

When trauma or effort damages the heart, cardiac symptoms, with rare exceptions, develop almost immediately. Precordial pain or breathlessness or both are common and compel the patient to stop whatever he is doing. There may be dizziness, fainting or shock, but these signs may be missing. Complete collapse need not occur and often, after a short rest, the patient is able to carry on for a while or is able to make his own way home. Immediate examination of the heart may not reveal any significant abnormalities. Warburg [3] reported that "fifteen of the fifty-one indubitable cases that were examined post mortem did not immediately after the trauma present such symptoms that there was reason to presume that the heart had been injured."

There is considerable variation in the time that elapses before the full symptoms and incapacity develop. In many instances extensive cardiac damage occurs immediately after the accident or follows within a few hours. In others there is a free interval of many hours or even of several days between the accident and the development of major cardiac damage, and during this interim the patient may complain of little beyond weakness and some uneasiness in the chest. In still other cases there are continuous symptoms of minor cardiac embarrassment after the accident, but these may not be severe enough to incapacitate the patient. Finally after days or even weeks a complete coronary occlusion occurs, leading to complete disability. Case 1 of my series is a good illustration of this sequence. Immediately on receiving the injury the patient had to rest for a while and then continued to work with difficulty. During the following ten days there developed progressively more severe symptoms of angina pectoris, but he still remained at work. Finally, on the eighteenth day after the accident, coronary thrombosis developed, causing him to remain bedridden for eight weeks. Although the major disablement was delayed eighteen days, there was a continuous train of symptoms between the time of the accident and the coronary occlusion.

To relate an accident or an unusual effort to a subsequent coronary thrombosis or syndrome of angina pectoris one must predicate cardiac symptoms accompanying or immediately following the event to which the cardiac injury is ascribed. If the symptoms are immediately disabling the causal connection is clear. If a lapse of days occurs between the causal event and the complete disablement there must be a continuity of symptoms dating from the event. It is conceivable that an intimal coronary hemorrhage might be caused by severe bodily strain and that it might give rise to no symptoms for several days, until marked obstruction of a coronary artery has taken place. Such a train of events is difficult to prove, and the possibility of its occurrence does not yet warrant its presentation as fact in a court of law.

It has been indicated that the occurrence of coronary thrombosis or the syndrome of angina pectoris after unusual effort gives presumptive evidence of antecedent sclerosis of the coronary arteries. Such arteriosclerotic lesions may give rise to no symptoms and to no cardiac disturbances; they may be nothing but evidence of normal aging. From the point of view of the law, the patient is not diseased nor has he clinical disease. When an unusual strain during work is followed by cardiac disability in the sense outlined, and when this occurs in a person who has been previously well and free from symptoms while at work, it is proper to conclude that the disability was induced by the work and is therefore compensable.

SUMMARY

In fourteen cases coronary occlusion or the syndrome of angina pectoris immediately followed a nonpenetrating injury to the precordium or unusual bodily effort. Eleven additional similar cases have been observed, and further cases have been reported in the literature.

Pathologic studies of several authors suggest the mechanism by which such traumatic lesions of the coronary circulation can be induced.

When a person previously free from symptoms of coronary artery disease has such symptoms after an injury to the chest or unusual physical exertion, the heart disease may be attributed to these external factors. When a person with simple angina pectoris has coronary artery occlusion following such injury or effort the sudden progression of the lesion may be ascribed to these causes. When such a sequence of events occurs in a person gainfully employed while he is at his task, it is proper to conclude that the cardiac disability is compensable, provided the cardiac symptoms accompany or follow immediately on the event to which the cardiac injury is ascribed and provided there is a continuity of symptoms dating from the event to the time of complete disability. Such disability may be delayed by several days or weeks.

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This Disruption of Medicine.—First physiology abandoned medicine, and in going took largely from medicine its function of studying the normal processes of the body; it also took something more important, for clinical medicine seemed to relax its claim to the use of the experimental method, which it had forged and which physiology, its offspring, now chose as its own chief weapon. Pathology has budded or is budding off, assuming for its part the study of the mechanism of disease, human or otherwise, and in almost all its aspects. This disruption of medicine, as it was originally constituted and as Harvey knew it, may be the inevitable outcome of its growth, may profit physiology, may profit general pathology, may even profit the medical sciences when these are viewed as one whole; but it is a process which can be carried too far, and it then becomes, is becoming, detrimental to the development of the clinical branches of work. It is necessary for medicine, still the parent and still largely possessed of guiding authority, to call a check to this subdivision of her estate.—Lewis, Sir Thomas: Research in Medicine and Other Addresses, London, H. K. Lewis & Co., Ltd., 1939.

BLOOD STUDIES IN BRUCELLOSIS

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In another paper [1] it was pointed out that the introduction of various intracutaneous tests and Huddleson's opsonocytophagic reaction, while providing a new approach to the study of brucellosis, has raised certain confusing problems which have not yet been solved. It seems probable that these tests, together with the agglutination reaction, are of service in indicating whether or not the tissues have been invaded by Brucella at one time or another, in this respect affording information of essentially the same value and limitations as the tuberculin test in suspected tuberculosis or the Wassermann test in syphilis. Doubt has properly been cast on the infallibility of Huddleson's diagnostic criteria, however, by the facts that brucellosis, a common and endemic disease, may be latent or asymptomatic and that the finding of positive tests may thus not indicate active disease but merely coincidental infection of no significance.

The difficulties are further increased by the paucity of physical signs, so that at the present time the diagnosis of brucellosis must rest on indirect laboratory evidence plus a more or less typical symptomatology. It becomes important, therefore, to search for measurable objective abnormalities sufficiently typical and frequent to assist in determining the presence or absence of activity. In a series of patients tested for brucellosis, we have encountered certain morphologic changes in both red and white blood cells so frequently as to warrant a somewhat detailed report.

METHODS AND MATERIALS

Source of clinical material and diagnostic methods were detailed in another communication.[1] Studies of the white blood cells are reported in 271 cases and of the red blood cells in 286 cases. Tests were made in all cases by Foshay's antiserum intracutaneous test,[2] by macroscopic agglutination tests read after forty-eight hours and by Huddleson's opsonocytophagic test (modification of R. M. C. described by Evans.[3]).

Huddleson [4] has stated that a positive cutaneous reaction (brucellergin) with low or absent opsonins indicates active disease. It is generally recognized, however, that cutaneous reactions in any disease are such delicate indicators of bacterial invasion that they may be positive even in asymptomatic disease and hence unreliable as indexes of activity. This objection likewise obtains in Foshay's antiserum test, for this reaction appears to furnish information of the same significance as those tests in which antigens or antigenic derivatives are employed. Frequently patients are encountered who present symptoms compatible with a diagnosis of chronic brucellosis and who yet give only a positive cutaneous reaction. In order to determine whether the hematologic abnormalities are as frequent in this group of cases as in those with more definite evidence of Brucella invasion, we have divided our cases into two series: Group I includes those patients with only a positive antiserum cutaneous reaction, opsonins and agglutinins being absent, while group II includes those patients who, in addition to a positive cutaneous reaction, also had demonstrable agglutinins (1:40 or higher) or opsonins (at least one cell out of twenty-five showing phagocytosis of twenty or more organisms).

Technical Procedures.—All determinations were made with Bureau of Standards glassware. Blood was collected by venipuncture and clotting was prevented by a mixture of potassium and ammonium oxalate (0.05 Gm. of ammonium oxalate and 0.04 Gm. of potassium oxalate to 5 cc. of blood). Counts of the white cells were made in both chambers of a double hemacytometer, the total counts on the two sides checking within 400 cells. Differential counts were done by the two coverslip method and stained with Wright's stain. In every instance 100 cells were classified on each of two separate coverslip preparations; if discrepancies were noted, the slips were discarded and the process was repeated.

TABLE 1.—*Differential Counts in Cases Showing Lymphocytic Leukocytosis*

Case	Group	Age	Total White Blood Cells	Segmented Polymorphonuclears	Stab Polymorphonuclears	Eosinophils	Basophils	Small Lymphocytes	Large Lymphocytes	Young Lymphocytes	Plasma Cells	Monocytes	Young Monocytes
867	II	40	10,300	39.5	10.5	2.5	1.0	3.5	28.5	4.0	...	10.5	...
897	II	73	11,000	38.0	13.0	1.0	1.0	8.5	31.5	1.0	...	6.0	...
968	I	42	11,600	33.0	5.0	2.5	0.5	4.0	37.0	10.0	...	8.0	...
914	I	28	9,900	46.5	5.0	...	0.5	5.5	35.5	2.0	...	5.0	...
848	I	7	11,100	44.5	6.5	2.5	...	15.0	23.5	3.5	...	4.0	0.5
846	I	4	10,100	49.0	2.0	0.5	1.0	7.0	30.0	1.0	...	9.0	0.5
843	I	5	11,200	30.5	4.5	...	0.5	20.5	32.0	5.0	...	7.0	...
748	I	47	11,500	32.5	7.5	3.0	0.5	16.5	21.5	3.5	1.5	13.5	...
695	I	35	9,900	45.0	8.0	2.0	...	23.0	17.0	2.0	...	3.0	...
972	II	60	12,700	52.5	3.5	4.0	1.5	4.0	27.0	1.5	1.0	5.0	...
947	II	39	12,600	40.5	2.5	3.5	0.5	2.0	31.0	10.0	0.5	9.5	...
893*	II	3	10,000	33.5	0.5	5.5	1.0	8.5	42.0	3.0	0.5	5.5	...
814	II	45	9,600	27.5	5.0	4.5	0.5	17.0	28.5	2.0	14.5	0.5	...
769	II	27	11,700	38.5	5.5	3.0	...	26.5	13.5	6.5	...	6.5	...
739	II	34	9,500	32.0	4.5	6.0	1.0	14.0	28.0	5.0	...	9.5	...
706	II	19	10,300	27.0	6.0	4.5	1.5	15.0	36.0	5.0	...	5.0	...
645	II	2	15,000	10.0	3.0	0.5	0.5	24.0	48.0	1.0	...	11.5	0.5
865	I	33	10,300	54.0	1.5	2.0	1.0	16.0	20.0	2.0	...	3.5	...

* The structure of the white cells in this case was strongly suggestive of infectious mononucleosis; but the heterophile antibody test was negative on two occasions.

In the erythrocyte studies, Wintrobe's [5] methods were employed throughout. Red blood cell counts were made from the same pipet on the two sides of a double hemacytometer, the two counts checking within 100,000 cells. Pipets were invariably shaken well in a mechanical shaker. Hemoglobin was determined by the Sahli method with instruments standardized both by the Bureau of Standards and by the Van Slyke method. In all charts dealing with erythrocytes, percentages rather than actual numbers of cases have been used, to permit comparison of our results with the normal range as determined by Wintrobe.[6]

THE LEUKOCYTES

It is generally stated that brucellosis is characterized by leukopenia with relative lymphocytosis. Amoss,[7] however, pointed out that the disease in many instances is actively lymphocytogenic, normal or increased total white blood cell counts often showing a high percentage

From the Brucellosis Laboratory, Clayton Foundation for Research.

1. Calder, R. M.: Chronic Brucellosis, South. M. J., to be published.
2. Foshay, Lee.: J. Infect. Dis. **59:** 330 (Nov.-Dec.) 1936.
3. Evans, Alice C.: Pub. Health Rep. **53:** 1507 (Aug. 26) 1938.
4. Huddleson, I. F.; Johnson, H. W., and Hamann, E. E.: Am. J. Pub. Health **23:** 917 (Sept.) 1933.
5. Wintrobe, M. M.: Am. J. M. Sc. **177:** 513 (April) 1929; Blood of Normal Young Women Residing in a Subtropical Climate, Arch. Int. Med. **45:** 287 (Feb.) 1930.
6. Wintrobe, M. M.: Bull. Johns Hopkins Hosp. **53:** 118 (Sept.) 1933.
7. Amoss, H. L.: Internat. Clin. **4:** 93 (Dec.) 1931.

of lymphocytes. In our experience acutely and seriously ill patients ordinarily have leukopenia. But in the chronically ill patients in this series leukopenia is not invariable (chart 1). The average total leukocyte count was found to be 7,225 in the group I cases, 7,130 in the group II cases and 7,173 in all cases. Counts above 9,500 not explainable on the basis of obvious complications occurred in thirty instances; of these, only twelve showed a polymorphonuclear leukocytosis while eighteen showed lymphocyte percentages of from 38 to 63 per cent (table 1).

The Lymphocytes.—Evidence of active stimulation of lymphopoietic centers was observed in almost every case. Two hundred and six (76 per cent) of the 271 patients had more than 30 per cent of lymphocytes, and

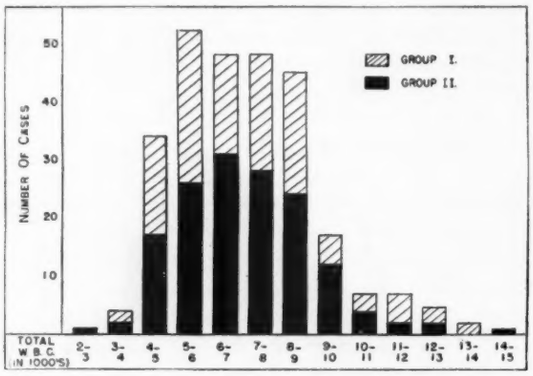

Chart 1.—Total leukocytes.

forty-five patients (16.6 per cent) had more than 50 per cent of lymphocytes (chart 2). Absolute numbers of lymphocytes were likewise increased; 151 of the 271 patients showed more than 2,500 of these cells, and in thirteen cases they numbered more than 5,000 (chart 3). Active lymphocytogenesis was further evidenced by the appearance of immature lymphocytes in the peripheral blood, as studied by the methods of Wiseman.[8] According to Wiseman an absolute value of approximately 100 is the usual maximum of these elements for the normal person. In contrast to this norm, four fifths of our patients showed values higher than 100. Wiseman's studies indicate that the occurrence of such forms constitute a "shift to the left" in the

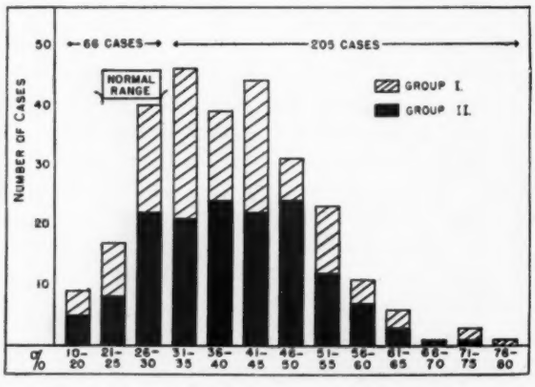

Chart 2.—Percentages of total lymphocytes.

lymphocyte series analogous in import to the well known "shift to the left" in the granulocyte series in the usual Schilling count (chart 4).

Microscopic Appearance of the Lymphocytes.—The mature lymphocytes in our cases frequently showed only a few or even no azurophilic granules. Large lymphocytes were more frequent than small lymphocytes, but this fact, as pointed out by Wiseman, is probably due to fixation and drying. Those cells having relatively large amounts of cytoplasm were not suggestive of the cells of infectious mononucleosis, in that nuclei of

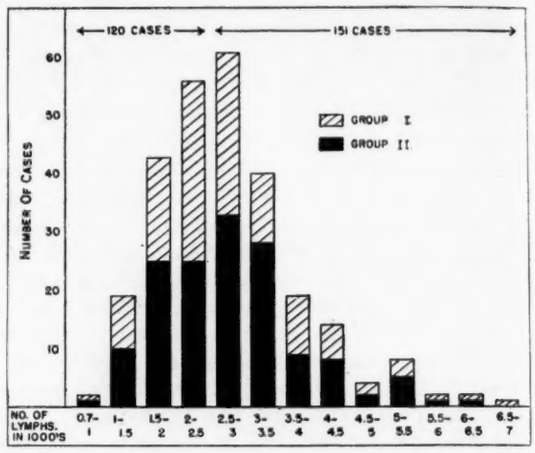

Chart 3.—Distribution of lymphocytes (absolute numbers) by cases.

ameboid shape were not encountered, nor was the cytoplasm of the foamy, light blue appearance characteristic of mononucleosis. Only one exception was noted, and in it the heterophile antibody test was negative on two occasions.

The immature lymphocytes were characterized by the frequent occurrence of nucleoli. The nucleus of these young cells was purplish blue and of fairly even distribution, and the cytoplasm, which contained no granules, was deeply basophilic, approaching true blast

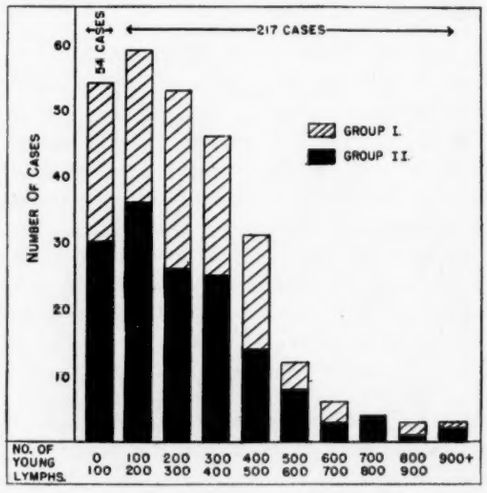

Chart 4.—Young lymphocytes (absolute numbers) by cases.

cells in its affinity for the basic stains. All gradations of basophilia were observed in different cells, but in our data only those cells with deeply basophilic cytoplasm were classified as immature.

Some of the cells, obviously immature because of their cytoplasmic basophilia, differed from those described in that their nucleus was small, round and intensely stained; their chromatin, while densely clumped, was not arranged in orderly fashion. Still other cells, which we classified as young lymphocytes, approached plasma cells in appearance, the chromatin being arranged in typical cart-wheel formation and the nucleus occasionally

8. Wiseman, B. K.: J. Exper. Med. 54: 271 (Aug.) 1931.

even being eccentrically placed. We did not classify this type as a plasma cell, however, because the cytoplasm seemed identical in appearance to that of the immature lymphocytes, and the cells were definitely smaller than typical plasma cells.

Plasma Cells.—In slightly more than one third of our cases, cells possessing all the characteristics of plasma cells were observed. When present, they accounted for from 1 to 3 per cent of the total leukocytes. It is not our purpose to discuss whether or not plasma cells belong to the lymphocytic series; in our tables they have not been so classified. If they actually belong in the

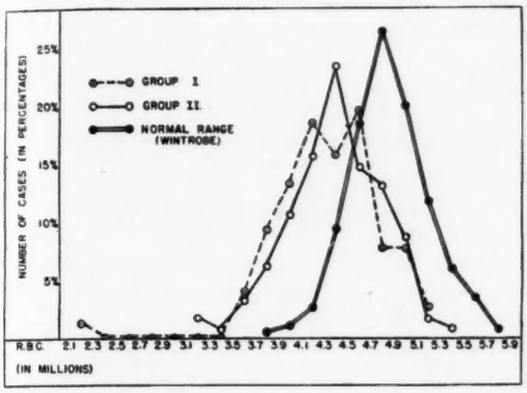

Chart 5.—Red blood cell counts (females).

lymphocytic series, their presence is additional evidence of the lymphocytogenic effect of Brucella infection.

The Monocytes.—In general, the absolute number of monocytes was not increased. Of the 271 cases, 227 (approximately 84 per cent) showed less than 700 monocytes, while forty-four (approximately 16 per cent) showed more than this number. Only seven patients had more than 1,000 monocytes, the maximum being 1,800; only one of these patients was a child, and in all of them tuberculosis had been excluded by appropriate tests.

Structurally there was nothing especially striking about the monocytes. We gained the impression that

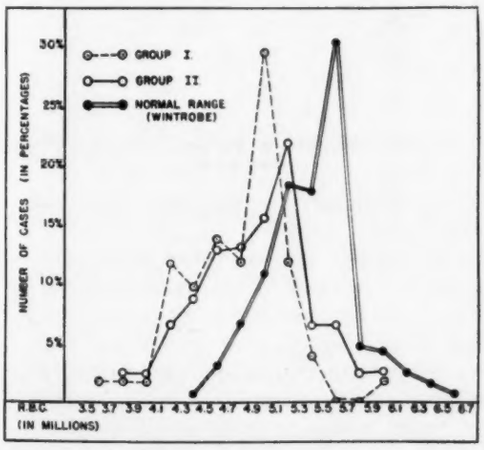

Chart 6.—Red blood cell counts (males).

more than the usual number of these cells showed diffuse, fine, pink granulation and that immature forms, as evidenced by basophilia of the cytoplasm, were more frequent than in normal bloods; but these changes were not definite enough to appear significant.

Monocyte-Lymphocyte Ratio.—Cunningham and Sabin and their associates[9] have stressed the importance of the monocyte-lymphocyte ratio as an index of the state of activity in tuberculosis. Since in many respects the fundamental pathologic changes of tuberculosis and brucellosis are similar, a determination of

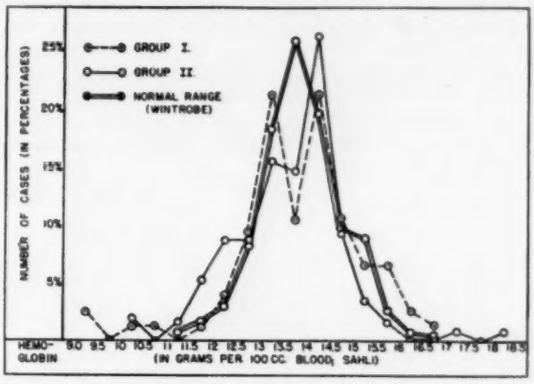

Chart 7.—Hemoglobin (females).

the monocyte-lymphocyte ratio in our cases is of interest. In almost all instances, the observed monocyte-lymphocyte ratio was lower than normal (0.33); it was normal or above in only twenty-seven cases. Extremely low values (0.15 or less) were observed in more than half of the cases. Attempts to relate the monocyte-lymphocyte ratio to state of activity, severity of infection or prognosis must await further observations.

The Granulocytes.—The total number of granulocytes was almost invariably reduced, both in percentages and in absolute numbers. The percentage of "stab" neutrophils was often elevated, but the increase in absolute numbers of these elements was not so striking. The left shift in certain cases is reminiscent of the counts in typhoid; but in our experience the percentage of "stabs" is rarely as high as in typhoid. It would appear from these observations that, in certain instances at least, myelogenic stimuli are present but that peripheral destruction exceeds their production. The question is of practical importance because of the phagocytic role of the polymorphonuclear cells in ridding the body of Brucella.

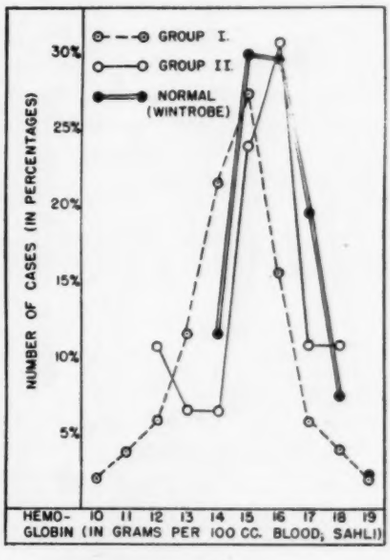

Chart 8.—Hemoglobin (males).

In one tenth of the cases, basophils were present in proportions greater than 1 per cent, the maximal percentage observed being 3. Approximately one fifth of these cases showed eosinophil counts of 5 per cent or more. Even excluding those cases for which there is an obvious explanation of the eosinophilia, the increase

9. Cunningham, R. S.; Sabin, Florence R.; Sugiyama, S., and Kindwall, J. A.: Bull. Johns Hopkins Hosp. **37**: 231 (Oct.) 1925.

in these cells is noteworthy. "Stab" eosinophils were frequent, at times equaling the mature forms in number. The significance of these observations is not apparent.

THE ERYTHROCYTES

Total Erythrocyte Counts.—As will be seen from charts 5 and 6, severe grades of anemia were rarely encountered. On the average, the red blood cell counts were approximately a half million lower than normal. Mean values for the various groups are shown in table 2. In the few cases showing severe grades of

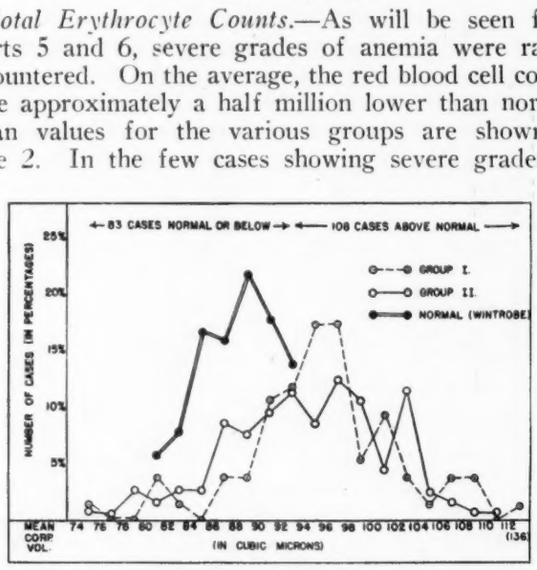
← 83 CASES NORMAL OR BELOW → ← 108 CASES ABOVE NORMAL →
GROUP I.
GROUP II.
NORMAL (WINTROBE)
NUMBER OF CASES (IN PERCENTAGES)
MEAN CORP. VOL.
(IN CUBIC MICRONS)

Chart 9.—Mean corpuscular volume (females).

anemia, an explanation of the anemia (menorrhagia, secondary infections, coincidental malaria, inanition and the like) was usually obvious.

Hemoglobin Determinations.—The values for hemoglobin, as determined by the Sahli method and calculated in grams per hundred cubic centimeters of blood, showed curves closely paralleling normal (charts 7 and 8). The average values (table 2) also approximated normal.

Volume of Packed Red Blood Cells.—On the average, the volume of the packed red blood cells (obtained by centrifuging in hematocrit tubes and noting the proportion of packed red cells to supernatant plasma) showed a smaller departure from normal than did the total erythrocyte counts (table 2).

TABLE 2.—*Summary of Mean Values*

	Group I Cases	Group II Cases	All Cases	Normal Mean (Wintrobe)
Red blood cells, female	4.32	4.39	4.37	4.85
Red blood cells, male	4.75	4.91	4.82	5.43
Hemoglobin, female	13.72	13.59	13.64	13.90
Hemoglobin, male	14.58	15.46	14.99	15.90
Volume of packed red blood cells, females	41.15	41.16	41.15	41.80
Volume of packed red blood cells, males	43.59	45.72	44.59	47.70
Mean corpuscular volume, females	95.80	94.20	94.80	88.00
Mean corpuscular volume, males	91.90	93.50	92.70	86.50
Mean corpuscular hemoglobin content, females	31.90	31.10	31.40	29.20
Mean corpuscular hemoglobin content, males	30.80	31.60	31.20	29.10
Mean corpuscular hemoglobin concentration, females	33.30	33.00	33.20	33.50
Mean corpuscular hemoglobin concentration, males	32.50	33.90	33.60	34.00

Mean Corpuscular Volume and Hemoglobin Content.—The disproportion between volume of packed red cells and total erythrocyte counts is accounted for by the fact that the average volume of the individual red cell is significantly greater than normal. Similarly, the disparity between total hemoglobin and erythrocyte counts is reflected in hyperchromia of the red blood cells. Charts 9, 10, 11 and 12 show these changes in detail. Definite macrocytosis (charts 9 and 10) was present in more than half of the cases, and the incidence of hyperchromia was likewise striking. In only six cases of apparently uncomplicated brucellosis were microcytosis and hypochromia observed.

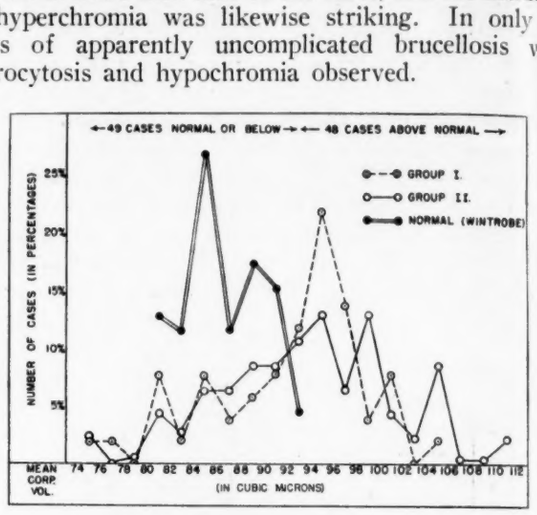
← 49 CASES NORMAL OR BELOW → ← 48 CASES ABOVE NORMAL →
GROUP I.
GROUP II.
NORMAL (WINTROBE)
NUMBER OF CASES (IN PERCENTAGES)
MEAN CORP. VOL.
(IN CUBIC MICRONS)

Chart 10.—Mean corpuscular volume (males).

As a check on the accuracy of our determinations of average erythrocyte volume, measurements of the diameters of red blood cells by the method of Price-Jones were made in twenty-three of these cases. By this method also a distinct macrocytosis is apparent (chart 13).

Mean Corpuscular Hemoglobin Concentration.—Bethell [10] has called attention to the fact that, if the plasma proteins are low, osmotic relationships between red cells and plasma are disturbed, with resultant swelling (relative macrocytosis) of the erythrocytes. Since the hemoglobin content of the cell remains constant, the swelling of the cell results in its being less saturated with hemoglobin than usual. Such a condition results in the triad of macrocytosis (cell volume greater than

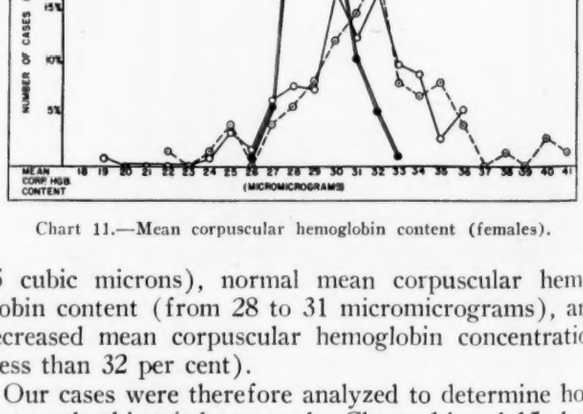

Chart 11.—Mean corpuscular hemoglobin content (females).

95 cubic microns), normal mean corpuscular hemoglobin content (from 28 to 31 micromicrograms), and decreased mean corpuscular hemoglobin concentration (less than 32 per cent).

Our cases were therefore analyzed to determine how frequently this triad occurred. Charts 14 and 15 show the hemoglobin concentrations of all patients studied. It will be noted that the hemoglobin concentration is low in a fairly large percentage of cases (sixty-eight cases). But these data include forty-five cases of definite hypochromia, most of them presenting complications sufficient to account for the low hemoglobin

10. Bethell, F. H., and Rottschaefer, Gerald: Univ. Hosp. Bull., Ann. Arbor **2**: 65 (Nov.) 1936.

content. Excluding these and two cases of pregnancy, there remain only twenty-one cases (7.3 per cent) in which the possibility exists that the observed macrocytosis was due, partly at least, to a circulatory phenomenon.

In contrast to this small number of patients in whom the macrocytosis was apparently merely a physical phenomenon, it is impressive to note the large number of cases in which the hemoglobin concentration of macrocytes was normal or even high. The conclusion seems justified that the observed macrocytosis is due not merely to physical alteration of osmotic relationships but to defects of maturation.

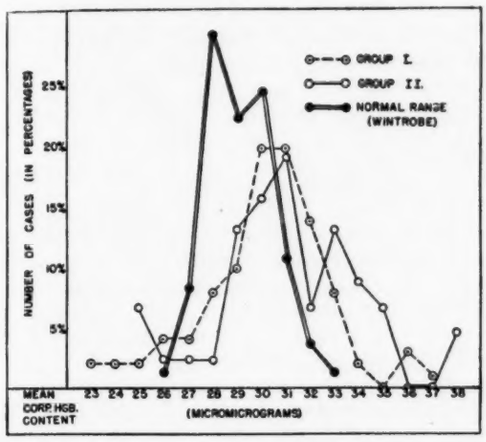

Chart 12.—Mean corpuscular hemoglobin content (males).

Microscopic Appearance of Erythrocytes.—Variations in size and shape of the erythrocytes were minimal. In general, the cells were all larger than normal and their hemoglobin content was likewise uniformly increased. Hypochromia and microcytosis were distinctly rare. In a very few instances pencil-shaped cells were observed. Nucleated red blood cells were infrequent. In two or three cases the inclusion of red blood cells in monocytes was observed. Polychromatophilia of slight degree was present in approximately 10 per cent of the cases.

Reticulocytes.—Reticulocyte counts were done in twenty-two of the cases reported in this series. The values ranged from 0.3 to 3.3 per cent, with mean values of approximately 1 per cent.

TABLE 3.—*Sedimentation Rates of Erythrocytes*

Millimeters per Hour	Group I Cases		Group II Cases	
	Male	Female	Male	Female
1-5	8	1	15	3
6-9	10	13	4	15
10-15	10	16	11	22
16-25	12	23	9	32
26-35	5	10	4	23
36-45	2	6	1	8
46-55	2	5	3	9
56-65	2	1	0	3

Coagulation.—In approximately one third of these cases, clotting was slow and incomplete. Free cells were often present even after the blood had stood overnight in the ice box. Normal retraction of the clot was rare; often there was no retraction at all. The coagulation defects do not seem to be related to decrease in blood platelets, for these elements are grossly normal in number and appearance in the stained preparations.

Sedimentation Rate of Erythrocytes.—Moderate acceleration of sedimentation rates (above Wintrobe's normal of 9 mm. for males and 15 mm. for females) occurred in slightly more than one third of these cases.

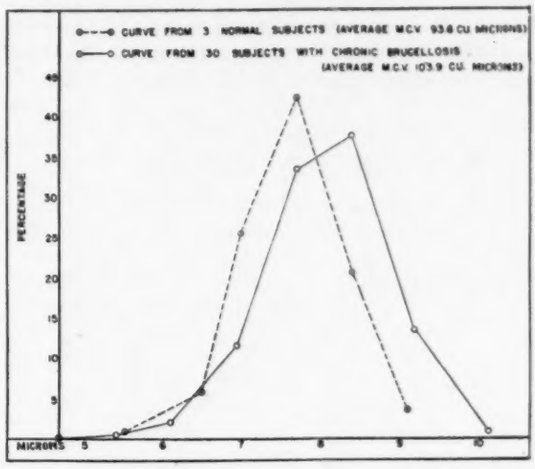

Chart 13.—Price-Jones curves in chronic brucellosis.

Very slow rates were not uncommon. Excessively fast rates were usually explainable on the basis of complications, such as arthritis or effusions (table 3).

Gastric Analysis.—Gastric analysis was done in ten cases in which there were macrocytosis and hyperchromia. Our procedure was to aspirate the fasting contents and then administer 50 cc. of 7 per cent alcohol and 0.4 mg. of histamine, specimens being withdrawn at fifteen minute intervals.

In nine of the ten cases there was no free hydrochloric acid in the fasting specimens. All of them, however, showed free acid after histamine. Values

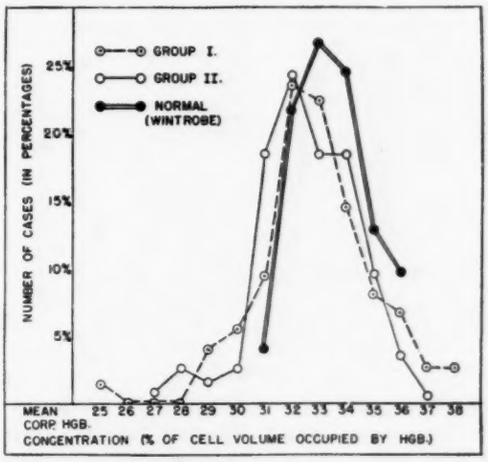

Chart 14.—Mean corpuscular hemoglobin concentration (females).

were definitely low in five cases, in the lower limits of normal in two cases and within the usual range of normal in three cases. Neither complete histamine anacidity nor, on the other hand, excessively high acidity was encountered.

Van den Bergh Tests.—The van den Bergh test was performed on 142 untreated patients. Of these, eighty-seven (61 per cent) showed bilirubin values of less than 0.5 mg. per hundred cubic centimeters of blood, while fifty-five (39 per cent) showed higher values. In the latter group, most of the values were less than 1 mg., the highest being 1.8 mg. The reaction was of the delayed direct type, the color change occurring after

thirty seconds and reaching its maximal intensity before the addition of alcohol and ammonium sulfate.

Evidence of Damage to the Liver.—It has been recognized previously that portal cirrhosis of the liver is often associated with macrocytosis. Wintrobe [11] has summarized the literature on the subject, and his description of the blood changes in diseases of the liver parallels our observations on the blood in these cases: "The anemia . . . is rarely severe. The macrocytosis affects the great majority of the red corpuscles, which show relatively little variation in shape or size. The lack of variation in the size of the corpuscles, however, does not distinguish the condition from pernicious anemia, for the distribution curves of the diameters of the red corpuscles are like those in cases of pernicious anemia with similar grades of anemia. Nucleated red corpuscles are uncommon and the fragility of the erythrocytes is normal." It is very unlikely that the cases which are included in our series represent the accidental association of pernicious anemia with brucellosis, for in the first place they are too numerous, and in the second place histamine anacidity has not occurred in those cases in which the gastric contents have been examined.

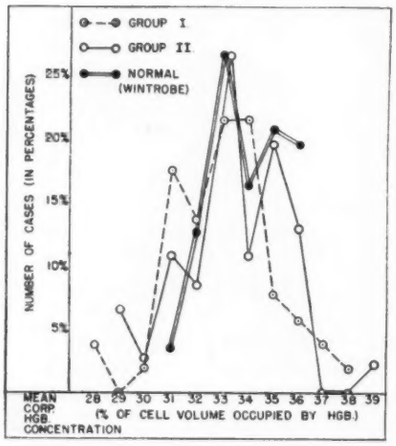

Chart 15.—Mean corpuscular hemoglobin concentration (males).

The striking parallelism between our observations and those cited by Wintrobe as occurring in hepatic disease makes reasonable the conclusion that these changes in all probability are due to anatomic or functional derangement of the liver. Contributory evidence for this idea is held in the facts that (1) low grade jaundice as measured by the van den Bergh reaction is not uncommon, (2) this reaction is of the delayed direct type, which ordinarily is associated with structural damage to liver parenchyma, and (3) the liver is, in our experience and that of others, more commonly enlarged in this condition than is the spleen.

SUMMARY

Structural studies of the blood were made in almost 300 patients with evidence of Brucella invasion. Normal leukocyte counts occurred in one half, leukopenia in one third and leukocytosis in one sixth of the cases. Active lymphocytosis was the most striking feature encountered and was evidenced by an increase in both percentage values and absolute numbers of lymphocytes and by unusually high numbers of immature lymphocytes (a lymphocytic "shift to the left"). Plasma cells were frequent. Monocytes in general were not increased in number, and the monocyte-lymphocyte ratio was almost invariably low. Many cases showed a moderate increase in percentage of "stab" neutrophils, but the total number of neutrophils was usually reduced. In one fifth of the cases there was moderate eosinophilia, often with immature forms.

Mild anemia of the macrocytic, hyperchromic type was frequent. Evidence was obtained that these changes are due to maturation defects, probably the result of damage to the liver.

Coagulation of the blood was slow and often incomplete. Clot retraction was also imperfect. The sedimentation rate was usually not extremely high except when complications, such as arthritis, were present.

These abnormalities of the blood were observed so consistently that they appear to represent a fundamental part of the disease with which these patients are suffering. So far as we are aware, they are duplicated in their entirety by no other known disease. It would seem, therefore, that the regularity with which they occurred in this group of patients is confirmatory evidence of the specificity of the tests on which the diagnosis of brucellosis was made.

Further analysis of our data indicates that these blood changes occur as often in patients with only a positive cutaneous reaction as in those who also show agglutinins and opsonins. Since these abnormalities seem to be a specific effect of Brucella invasion, their equal incidence in the two groups of patients indicates that, in many instances, a positive antiserum cutaneous reaction may be the sole evidence of brucellosis.

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11. Wintrobe, M. M.: Relation of Disease of the Liver to Anemia, Arch. Int. Med. **57**: 289 (Feb.) 1936.

PLASMA PROTHROMBIN AND THE BLEEDING TENDENCY

WITH SPECIAL REFERENCE TO JAUNDICED PATIENTS AND VITAMIN K THERAPY

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It is our main purpose in the present article to discuss treatment of the bleeding tendency so often seen in patients suffering from disease of the biliary tract. Bleeding from mucous surfaces often occurs spontaneously in these patients, but the greatest danger is from bleeding at operation or from the wound after operation.

Through the work of several laboratories [1] it has been shown that a newly discovered vitamin, vitamin K,

From the Mercy Hospital and from the Department of Pathology, State University of Iowa.
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1. Reported by:
Dam, Henrik: The Antihemorrhagic Vitamin of the Chick, Biochem. J. **29**: 1273 (June) 1935.
Dam, Henrik; Schønheyder, Fritz, and Tage-Hansen, Erik: Studies on the Mode of Action of Vitamin K, ibid. **30**: 1075 (June) 1936.
Schønheyder, Fritz: The Quantitative Determination of Vitamin K, ibid. **30**: 890 (May) 1936.
Almquist, H. J., and Stokstad, E. L. R.: Hemorrhagic Chick Disease of Dietary Origin, J. Biol. Chem. **111**: 105 (Sept.) 1935.
Quick, A. J.: The Coagulation Defect in Sweet Clover Disease and in the Hemorrhagic Chick Disease of Dietary Origin, Am. J. Physiol. **118**: 260 (Feb.) 1937.
Hawkins, W. B., and Brinkhous, K. M.: Prothrombin Deficiency the Cause of Bleeding in Bile Fistula Dogs, J. Exper. Med. **63**: 795 (June) 1936.
Greaves, J. D., and Schmidt, C. L. A.: Nature of the Factor Concerned in Loss of Blood Coagulability of Bile Fistula Rats, Proc. Soc. Exper. Biol. & Med. **37**: 43 (Oct.) 1937.
Warner, Brinkhous and Smith.[2]
Butt, Snell and Osterberg.[3]
Dam and Glavind.[4]
Smith, Warner, Brinkhous and Seegers.[16]

plays an important part in prothrombin formation in the body. Patients with obstructive jaundice or with biliary fistulas, having no bile in the intestine, have difficulty in absorbing such fat-soluble materials. Without adequate absorption of vitamin K they suffer from faulty formation of prothrombin, and for this reason a tendency to bleed develops.

The therapeutic use of vitamin K in the treatment of jaundiced bleeders was first reported by Warner, Brinkhous and Smith [2] and almost simultaneously by Butt, Snell and Osterberg.[3] Additional cases were reported almost at once by Dam and Glavind.[4] All these reports have indicated that dramatic relief from the bleeding tendency is obtained in most cases by the administration of vitamin K. Further details of the literature on this vitamin may be obtained from the articles just cited.

BLEEDING IN OBSTRUCTIVE JAUNDICE: REPORT OF CASE

E. W., a white woman aged 53, who entered Mercy Hospital April 20, 1938, because of pruritus and jaundice, for the past two years had had periodic attacks of pain in the right upper quadrant of the abdomen. The attacks lasted from one hour to several days. Early in February they increased in severity and were accompanied by lassitude and weakness. Jaundice made its appearance at this time, but the patient refused hospital treatment until the pruritus had become intolerable.

She was well developed and well nourished. The skin showed marked jaundice and much excoriation because of the pruritus. There were considerable rigidity and tenderness in the right upper quadrant, but the gallbladder could not be felt. The temperature, pulse rate and respiratory rate were within normal limits. The stools were clay colored and the urine deep yellow. The coagulation time was six minutes by the capillary tube method.

At operation, April 22, a number of stones were removed from the gallbladder and one large stone was removed from the common bile duct. A drainage tube was placed in the gallbladder and another in the common bile duct.

After operation the patient showed no untoward symptoms for several days. The temperature remained at about 37 C. (98.6 F.). April 26 bile ceased to drain and some blood dripped from the tube. Irrigation of the tube was attempted but was discontinued because of pain. On the following day the dressings were found to be saturated with blood. The patient vomited 300 cc. of fluid which contained bright red blood. The stools were tarry black. Bleeding continued on the following day and the jaundice, which had been subsiding, began to increase, probably because of obstruction by blood clots. April 29 an attempt was made to give an enema, but before much fluid had been injected the patient expelled 1,000 cc. of reddish black fluid, obviously blood.

A sample of blood drawn at this time showed deep jaundice of the plasma (van den Bergh test, 17 mg. per hundred cubic centimeters). The coagulation time was thirteen minutes by the capillary tube method. The bleeding time (Duke's method) was greatly prolonged, the patient continuing to bleed for more than six hours from a simple stab wound. The red cell count, previously normal, had now fallen to 2,000,000. A transfusion of 300 cc. of blood was given April 29 and another April 30. These transfusions seemed to have little effect, for oozing from the operative wound continued.

At this stage, analyses for prothrombin carried out for the first time showed the plasma prothrombin content to be 8 per cent of normal, a result considered adequate to explain the bleeding. April 30 and May 1 vitamin K emulsified in 30 cc. of 2 per cent sodium taurocholate was given by mouth. Each dose contained the vitamin which had been extracted [4a] from 200 Gm. of dry alfalfa meal. Bleeding from the operative wound ceased within twenty-four hours of the onset of treatment. The treatment itself produced some nausea; hence the vitamin was given in capsule form on the following two days. May 3, after three days of treatment, the plasma prothrombin content had risen to 69 per cent of normal from the original 8 per cent. The bleeding time (Ivy's [5] method) was now six minutes. May 3 and 4 the vitamin, given in emulsion form in half the original dose, produced no nausea. During the following three days no vitamin K was given and May 7 the dressings again showed blood. Blood drawn for analyses showed that the plasma prothrombin content had fallen to 47 per cent, a level which we have come to consider in the upper part of the danger zone. From May 9 to 18 the patient received vitamin K emulsion equivalent to 100 Gm. of alfalfa meal daily. The bloody drainage ceased on May 10, and the plasma prothrombin content on May 17 was found to be 71 per cent of normal.

The jaundice increased for a few days after removal of the drain on May 1 but thereafter it subsided and on May 12 the van den Bergh test showed that the plasma bilirubin content had fallen to 4.1 mg. per hundred cubic centimeters. May 18 the jaundice had completely subsided and vitamin K therapy was discontinued. The remainder of the patient's stay in the hospital was entirely uneventful, except for a transient period of vomiting. The patient left the hospital June 5 and when seen August 23 had remained free from symptoms.

This case illustrates postoperative bleeding in a patient with chronic biliary obstruction. There was no serious bleeding at the time of operation, but four days after operation blood began to ooze at the operative site and this bleeding continued despite two transfusions. The plasma prothrombin concentration at that time was 8 per cent of normal. We have noted in a number of cases that when the plasma prothrombin level is low prior to operation one may anticipate a further fall after operation. This fall is presumably due to the utilization of prothrombin in forming fibrinous exudate at the operative site. The level at which bleeding actually occurs is variable, and the actual onset of bleeding is often precipitated by the operation itself or even by minor traumatic factors. Rarely does bleeding occur until the prothrombin has been reduced at least to 50 per cent of normal. Occasionally a level of 20 per cent will be tolerated without hemorrhage, but patients below the 50 per cent level are definitely in the danger zone.[5a]

Relief from bleeding in this case occurred within twenty-four hours after the institution of vitamin K therapy. This is in accord with previous experience [6] that the plasma prothrombin level rises rapidly out of the danger zone when vitamin K is absorbed in adequate amounts. At the end of three days of treatment the prothrombin level had risen to 69 per cent of normal, whereas it had been 8 per cent of normal. This case also illustrates the point that treatment should not be relaxed too early, for when it was discontinued the prothrombin level fell to 47 per cent within three days and bleeding began again. On resumption of therapy, bleeding again ceased and the prothrombin level rose to 71 per cent. One cannot urge too strongly that

2. Warner, E. D.; Brinkhous, K. M., and Smith, H. P.: Bleeding Tendency of Obstructive Jaundice: Prothrombin Deficiency and Dietary Factors, Proc. Soc. Exper. Biol. & Med. **37**: 628 (Jan.) 1938. Brinkhous, Smith and Warner.[4a]

3. Butt, H. R.; Snell, A. M., and Osterberg, A. E.: The Use of Vitamin K and Bile in Treatment of the Hemorrhagic Diathesis in Cases of Jaundice, Proc. Staff Meet., Mayo Clin. **13**: 74 (Feb.) 1938.

4. Dam, Henrik, and Glavind, Johannes: Vitamin K in Human Pathology, Ugesk. f. læger. **100**: 248 (March 10) 1938; Lancet **1**: 720 (March 26) 1938.

4a. Brinkhous, K. M.; Smith, H. P., and Warner, E. D.: Prothrombin Deficiency and the Bleeding Tendency in Obstructive Jaundice and in Biliary Fistula: Effect of Feeding Bile and Alfalfa (Vitamin K), Am. J. M. Sc. **196**: 50 (July) 1938.

5. Ivy, A. C.; Shapiro, P. F., and Melnick, P.: The Bleeding Tendency in Jaundice, Surg., Gynec. & Obst. **60**: 781 (April) 1935.

5a. Warner, Brinkhous and Smith.[2] Brinkhous, Smith and Warner.[4a]

6. Warner, Brinkhous and Smith.[2] Butt, Snell and Osterberg.[3] Dam and Glavind.[4]

therapy be continued at least as long as bile continues to drain at the operative site. This use of the vitamin should be extended to include the preoperative period. Had this particular patient been given vitamin for several days prior to operation, there is little doubt that excessive bleeding would have been prevented altogether.

PROTHROMBIN VALUES IN OTHER CONDITIONS

The accompanying table shows prothrombin values in a number of clinical conditions. In three cases of thrombopenia the plasma prothrombin values were within normal limits. These cases throw doubt on the view, occasionally cited,[7] that platelets are the source of prothrombin.

In three cases of hemophilia the prothrombin values were normal. In case 4 two weeks treatment with vitamin K produced no change in the prothrombin or the clotting time (forty-five to seventy-five minutes). This is in accord with the experience of Dam and Glavind.[4]

Miscellaneous Plasma Prothrombin Values

Case No.	Sex	Age (Yr.)	Diagnosis	Prothrombin, %
1	♀	13	Thrombopenic purpura	90
2	♂	0.5	Thrombopenic purpura	79
3	♂	31	Persistent thrombopenia, nonsymptomatic	100
4	♂	25	Hemophilia	96
5	♂	11	Hemophilia	86
6	♂	44	Hemophilia	92
7	♂	6	Acute leukemia	66
8	♂	46	Acute leukemia	82
9	♂	7	Acute leukemia	86
10	♂	55	Laënnec's cirrhosis; primary carcinoma of liver (autopsy)	79
11	♂	55	Laënnec's cirrhosis (clinical)	42
12	♂	24	Laënnec's cirrhosis (autopsy)	72
13	♂	29	Laënnec's cirrhosis (clinical)	66
14	♂	25	Laënnec's cirrhosis (autopsy)	30
15	♂	23	Laënnec's cirrhosis, slight; "Banti's disease" (laparotomy)	66
16	♀	50	Syphilitic "Banti's disease" (clinical)	65

In three cases of acute leukemia there was moderate reduction in the prothrombin level.

In six cases in which the diagnosis of Laënnec's cirrhosis of the liver had been made there was a reduction in the prothrombin level. In two of these the value was definitely in the danger zone, but in neither did we observe areas of bruising or evidence of bleeding from mucous surfaces, such as one sometimes sees in patients with this disease. Patient 11 was treated for two weeks with vitamin K, but the prothrombin level remained constant at about 40 per cent. Patient 16, with a clinical diagnosis of syphilitic Banti's disease, was treated in a similar manner but without effect on the prothrombin concentration. Such results indicate that the lowering of the prothrombin concentration is not the result of a deficiency in vitamin K.

COMMENT

There is as yet no proof that vitamin K is a component of the prothrombin molecule. It may be, instead, in some way essential to proper function of the tissues concerned in the manufacture of prothrombin.

Work from this laboratory indicates that the liver is vitally concerned in the process of manufacturing prothrombin, for the plasma prothrombin level falls when the liver is partially excised [8] or when it is injured by chloroform poisoning.[9] It seems probable that the lowering of the prothrombin level in cases of cirrhosis is due mainly to defective hepatic function, which is not the result of deficiency in the supply of vitamin K. This view is in accord with the fact, already reported,[4a] that patients with disease of the biliary tract may respond poorly to vitamin K if the liver is badly damaged (i. e. by obstructive biliary cirrhosis or by extensive growth of a neoplasm within the liver). It is now clear that prothrombin formation can be interrupted either by a reduction of the supply of vitamin K or by interference with the integrity of the liver. The vitamin is of therapeutic value in the former case.

The technic for titration of prothrombin is still somewhat too involved for general use and we have had little success in predicting bleeding by the use of tests of the bleeding time and clotting time. Many patients have normal values by these tests, and yet persistent bleeding develops. Unless prothrombin analyses are made, it is advisable to give the vitamin in a routine manner for several days to all patients with biliary disease both before operation and during the crucial postoperative period.[10] The vitamin appears to be entirely harmless, for we have given it to animals in doses many times as great as the amount recommended for therapeutic use.

It has been suggested that vitamin D may be useful in the treatment of the bleeding tendency of patients with disease of the biliary tract. Being fat soluble, this vitamin too is absorbed with difficulty when bile is absent from the intestine.[12] McNealy, Shapiro and Melnick [13] analyzed a large series of cases of disease of the biliary tract both with and without supplements of this vitamin. Their conclusions, based partly on clinical evidence and partly on tests of bleeding time, indicated a beneficial effect. A little later Boys [14] reached the same conclusion. Wigodsky and Ivy [15] have recently analyzed a vitamin K preparation and found it to contain so little vitamin D that its activity in such patients cannot be ascribed to the latter. It is possible that in human beings, when the disease is complicated by infection, a deficiency in vitamin D may contribute to the difficulty in forming prothrombin. However, our own experiments on dogs with sterile biliary fistulas [16] failed to show that vitamin D has any effect in raising the prothrombin level. In the aforementioned cases bile or bile salt was fed along with

7. Bayne-Jones, Stanhope: The Presence of Prothrombin and Thromboplastin in the Blood Platelets, Am. J. Physiol. **30**: 74, 1912.

8. Warner, E. D.: Plasma Prothrombin: Effect of Partial Hepatectomy, J. Exper. Med. **68**: 831 (Dec.) 1938.

9. Smith, H. P.; Warner, E. D., and Brinkhous, K. M.: Prothrombin Deficiency and the Bleeding Tendency in Liver Injury (Chloroform Intoxication), J. Exper. Med. **66**: 801 (Dec.) 1937.

10. Since this article was written a simple bedside test for control of vitamin K therapy has been devised by S. E. Ziffren, C. A. Owen and G. R. Hoffman of this laboratory and will be published in the near future. In brief, freshly drawn blood is mixed with one tenth its volume of thromboplastin [9] and the clotting time compared with that of a normal control. From the ratio of the two clotting times one can calculate the clotting power in percentage of normal. This test is considerably simpler than either our prothrombin method or the test advocated by Quick (Am. J. Physiol. **114**: 282 [Jan.] 1936).

11. Footnote deleted on proof.

12. Tammann, H.: Ueber die Beeinflussung der porotischen Osteomalazie nach Gallenfistel durch das D-Vitamin, Beitr. z. klin. Chir. **142**: 83, 1928. Greaves, J. D., and Schmidt, C. L. A.: The Role Played by Bile in the Absorption of Vitamin D in the Rat, J. Biol. Chem. **102**: 101 (Sept.) 1933.

13. McNealy, R. W.; Shapiro, P. F., and Melnick, P.: The Effect of Viosterol in Jaundice, Surg., Gynec. & Obst. **60**: 785 (April) 1935.

14. Boys, Floyd: A Report on the Value of the Ivy Bleeding Time Test and the Use of Viosterol in Cases of Obstructive Jaundice, Surgery **2**: 817 (Dec.) 1937.

15. Wigodsky, H. S., and Ivy, A. C.: Assay of a Vitamin K Preparation for Vitamin D, Proc. Soc. Exper. Biol. & Med. **38**: 785 (June) 1938.

16. Smith, H. P.; Warner, E. D.; Brinkhous, K. M., and Seegers, W. H.: Bleeding Tendency and Prothrombin Deficiency in Biliary Fistula Dogs: Effect of Feeding Bile and Vitamin K, J. Exper. Med. **67**: 911 (June) 1938.

vitamin D. Since the diet normally contains small quantities of vitamin K, it is possible that the bile salt given permitted enough absorption of this vitamin to raise the prothrombin level out of the bleeding zone. It is to be hoped that studies of human beings will be repeated with the plasma prothrombin level as a control on the procedure.

CONCLUSIONS

Our case illustrates postoperative bleeding in a patient with obstructive jaundice. Such bleeding is due to profound reduction in the plasma prothrombin level. When the patient was treated with vitamin K, the prothrombin level rose and bleeding ceased.

Patients with thrombopenic purpura and patients with hemophilia have normal prothrombin values, and evidence indicates that such patients obtain no relief from administration of vitamin K.

In three cases of acute leukemia the prothrombin level was found to be slightly reduced.

In cases of Laënnec's cirrhosis the prothrombin level may be markedly reduced, but evidence indicates that the reduction is due to defective hepatic function, which is not the result of deficiency in vitamin K.[17]

ACUTE INTERSTITIAL PNEUMONITIS: A NEW DISEASE ENTITY

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The Cornell University Infirmary receives practically all seriously ill patients from the student body. Minor ailments, such as common colds without fever, are taken care of at the medical adviser's office or in the offices of practicing physicians of the town.

The infirmary records of the twenty years from 1917 to 1937 reveal that (1) the many cases of "cold and fever" occurred irregularly through every year but always reached their peak sometime in December, January, February or March, (2) pandemic influenza occurred in 1918-1919 and 1919-1920 only, (3) interpandemic influenza occurred in definite outbreaks in 1925-1926, 1927-1928, 1931-1932 and 1936-1937 (table 1) and (4) from eight to sixteen cases of pneumonia occurred through each nine month college year (table 1).

TABLE 1.—*Diseases of the Respiratory Tract from 1925 to 1937*

Year	Student Registration	Cases of Uncomplicated Interpandemic Influenza	Cases of Interpandemic Influenza Complicated by Pneumonia	Cases of Pneumonia Unrelated to Influenza
1925-1926	5,132	644	4	4
1927-1928	5,205	437	3	11
1931-1932	5,916	639	3	13
1936-1937	6,177	824	4	8

In October 1937 there began to appear a type of infection of the respiratory tract different from anything that we had seen previously. It did not belong to any of the four categories listed. It did not attack large numbers, as interpandemic influenza had usually done, and it was not accompanied in the majority of cases by the bone ache and backache of typical influenza; it was apparently a new acute disease of the respiratory tract having as its cardinal feature specific lesions in the lungs inaudible to the stethoscope but definite in the roentgenogram of the chest. To this disease we have applied the term acute interstitial pneumonitis.

TABLE 2.—*Diseases of the Respiratory Tract from 1937 to 1939*

Year	Student Registration	Cases of Uncomplicated Interpandemic Influenza	Cases of Interpandemic Influenza Complicated by Pneumonia	Cases of Pneumonitis	Cases of Pneumonia Unrelated to Influenza
1937-1938	6,115	129	1	50	8
1938 to Jan. 24, 1939	6,569	41	0	27	4

Since October 1937, in no month when the infirmary was open have we failed to have cases of pneumonitis. There have been eighty-six cases as shown by definite radiographic evidence (table 2).

RADIOGRAPHIC EVIDENCE

Several of the patients had roentgenograms taken before definite evidence was visible on the film and were then observed almost daily as the pathologic change was developing in the lungs. Others were rechecked at regular intervals during the entire period of their illness. A review of the roentgenograms leads to the conclusion that (*a*) definite x-ray evidence may not appear until from thirty-six to forty-eight hours after the clinical onset, (*b*) the infection spreads from the hilus outward, following the bronchial and vascular tree, as do many other pulmonary infections, and (*c*) the cases can be roughly divided according to the radiographic evidence into three groups.

The onset has been marked by increased density in the hilar shadows, with a fan-shaped localized accentuation of and a numerical increase in the linear pulmonic markings extending from the hilus into the adjacent lung field. This extension is down into the base in the majority of the cases; it may be lateral or, not infrequently, into the first and second interspaces. In one group of cases, those of milder involvement, there then appeared over this area a diffuse even increase in density, which was not sufficient, however, to eliminate the linear markings and suggested an intense congestion rather than a true consolidation of lung tissue. The visible changes did not usually extend to the margin of the lung, and they cleared rather rapidly, ordinarily in from one to two weeks (fig. 1).

In a second group of cases, of more serious involvement, the infiltration in the fan extended more frequently to the periphery of the lung, with irregular areas of markedly increased density suggestive of lobular involvement and more typical of definite bronchopneumonia. Resolution was correspondingly slower than in the first group (figs. 2 and 3).

In about 5 per cent of the cases the involvement was severe. The development was similar to that in the preceding groups except that, in addition to the original focus, there was a diffuse spread throughout the remainder of the lung with partial or complete involvement of the other lung. This additional involvement was characterized by a generalized swelling of the pulmonic markings with a light mottling somewhat

17. Since this article was written, vitamin K has been placed on the market by Abbott Laboratories, North Chicago, Ill. The vitamin K preparations used in our own work were prepared partly by ourselves [4a] and partly on an experimental basis by the Upjohn Company of Kalamazoo, Mich.

From Cornell University Infirmary.

similar to that of pulmonary edema or intense passive congestion and additional irregular blotchy areas of very lightly increased density. We believe the underlying pathologic change in this type of case to be a definite interstitial inflammation. In this group there was a decided tendency to some involvement of the interlobar pleura and, in one case, of the parietal pleura. Clearing of the lung fields was slow, taking from one to two and one-half months (figs. 4, 5 and 6).

Fig. 1 (case 1).—A typical rounded fan present in the cases of mild involvement and frequently in the early stages when the disease is severe, showing the intensified linear markings through a hazy background.

COURSE

The onset of the disease is usually not sudden but is ushered in by a period of weakness, malaise and slight fever lasting for two or three days. The temperature gradually mounts, and the patient begins to complain of cough, pains in the chest or sore throat or a combination of these symptoms. (In some cases, however, the onset has been sudden, as in pneumonia.) At this stage there are usually signs of pharyngitis or a cough and a temperature of from 100 to 102 F., but stethoscopic examination of the chest gives no evidence of consolidation and there is little or no sputum. There may be a few fine rales. The radiographic evidence at this stage is usually that of group 1, and in many cases the disease recedes from this point (fig. 1).

As the disease progresses, as it does in cases of group 2, the temperature rises to 103 or 104 F., not infrequently exhibiting the peculiarity of being up in the morning and down in the evening. The patient feels quite ill at this stage, not infrequently complains of tightness in the chest and pain on coughing and sometimes shows the flushed cheeks so often seen with pneumonia. Stethoscopic examination of the chest still reveals no pleurisy or consolidation; there may be a few fine rales, and sputum is entirely lacking or scanty and has been in none of our cases either of the "rusty" or of the "cherry juice" type. Roentgenograms of the chest at this period show the fan-shaped area of increased density, generally extending to the periphery of the lung (fig. 3).

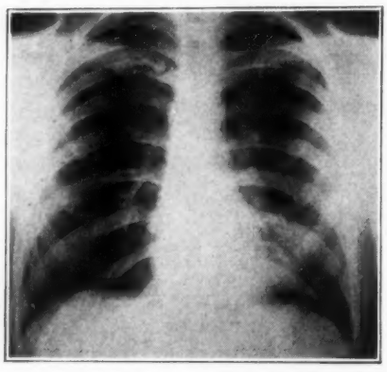

Fig. 2 (case 2).—A typical rounded basal fan on the second day of illness and the forerunner of a more severe infection (see fig. 3).

In our third group of cases, the onset and development of the disease was similar to that in group 2 except that there was a characteristic sudden jump in the temperature and evidences of toxicity, indicating a sudden and pronounced spread of the infection. The febrile period was more prolonged and more severe. Moist rales were invariably present, but the sputum was still scanty in proportion to what one might expect from the rales. The toxicity of the disease was severe, and the prognosis in each case for a number of days was grave. We believe that these cases are similar to those described by Dr. Hobart Reimann.[1]

The leukocyte counts have shown mild leukocytosis, the commonest count early in the disease being about 8,500 and running later up to 14,000 or 16,000. In one instance late in the disease there was a leukocyte count of 22,000. The percentages of polymorphonuclears has been in most instances between 63 and 75. In only one instance did it reach 88.

The pneumonitis terminates by lysis; there is no spectacular crisis. The patient was usually hospitalized ten days, though one of the four, with severe extension of the process to both lungs, required seventy-six days. Sixty-one of the eighty-six were hospitalized for a period not exceeding fourteen days. Complications are apparently rare, there having been no bronchiectasis, no organization of exudate and no abscess in our first eighty-six cases of the disease. There have, however, been evidences of mild pleuritic involvement in those of severe involvement. In the cases of rather rapid recovery in which the pulmonary lesions have previously been lacking, it is not unusual to find, after the patient has had a normal temperature for three or four days and been discharged from the infirmary, many coarse rales over the areas shown by roentgenogram to be involved. These rales may be present for as long as two weeks after the termination of treatment in bed. Though the course of the disease is comparatively short in the cases of mild involvement, it has not infrequently caused the loss of from 16 to 18 pounds (7 to 8 Kg.) in otherwise healthy young college students, and several patients have found themselves so weak at the termination of the disease that they have had to ask leave of absence for the remainder of the term to recuperate. That some students can take this disease in their stride is evidenced, however, by the fact that one of our football players had a typical attack in October 1938, recovered completely and went on to participate successfully in a hard schedule of intercollegiate football.

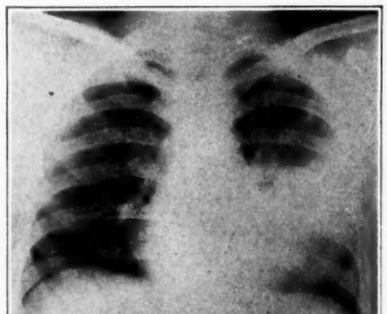

Fig. 3 (case 2).—Appearance on the eighth day of illness. The area of density has extended to the periphery of the lung and is considerably widened. This represents the maximum spread of the process in this case.

EPIDEMIOLOGY AND ETIOLOGY

Among the eighty-six patients so far observed, there have been six instances of two from the same house, one instance of five from the same house and one

1. Reimann, Hobart A.: An Acute Infection of the Respiratory Tract with Atypical Pneumonia, J. A. M. A. **111:** 2377-2384 (Dec. 24) 1938.

instance of four from the same house. In the clusters of four and five cases, the original patient was ill and coughing about the fraternity house for several days before he went to the infirmary; the other patients followed at intervals of from five to fourteen days in one instance and from thirteen to fourteen days in the other. The other patients admitted having gone into the room of the original patient and having talked with him, thinking he "just had a cough."

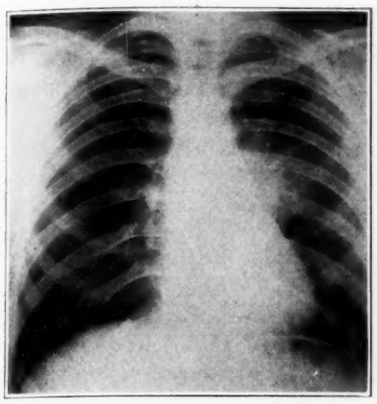

Fig. 4 (case 3).—Initial rounded fan on the third day of illness.

In the absence of cough, the infectiousness of the disease may not be very great, since we had in the series one instance in which, under the crowded conditions of a house party week-end, two students slept in the same bed with another student ill with pneumonitis (but not coughing) and remained well. A student who sat next to one with pneumonitis (who was coughing) in his economics class came down with the same disease twelve days later.

As a result of our limited experience, we feel that this disease is more infectious than pneumonia, and we now, as a routine, hospitalize and isolate all our patients, permitting no visitors.

In the absence of sputum, cough swabs have been obtained by tickling the pharynx. Cultures of material from these swabs and of sputum have frequently been reported as showing mixed Streptococcus viridans and Staphylococcus aureus. In mouse tests of the sputum at the Tompkins County laboratory, the mice have but rarely succumbed, but when they have, pneumococci have usually been found. Material sent to the New York State Health Department laboratory has led to occasional reports of the higher types of pneumococci, e. g. type VIII, types XV and XVIII and type XI. On the basis of evidence accumulated to date, no definite statement can be made as to cause, but a virus would seem more probable than bacteria.

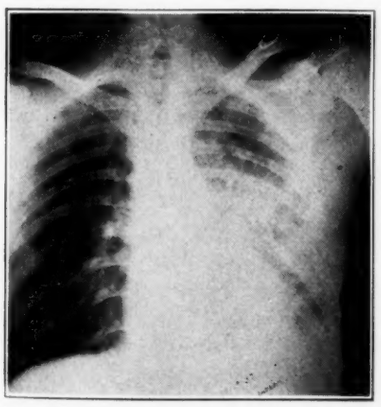

Fig. 5 (case 3).—Maximum spread on the ninth day of illness. Note the extension of the original fan and the diffuse mottling of the remainder of the originally involved lung and also of the other lung.

COMMENT

The question may well be asked Why is this disease not simply atypical pneumococcic pneumonia? We feel that it is not for the following reasons:

1. The areas of density shown in the roentgenograms are not lobar and are less dense than in the case of lobar pneumonia. The radiographic evidence in the majority of cases does not conform with that usually found in cases of bronchopneumonia. 2. The fever is intermittent rather than continuous, as in pneumonia. 3. There is milder leukocytosis and a smaller rise in the polymorphonuclear count than is the rule in pneumonia. 4. Severe sweating is much more common than in pneumonia. 5. The disease appears to be more communicable than pneumonia usually is. 6. The respiratory rate is not as high as that usually found with pneumonia. 7. There has been much less sputum and no "rusty" sputum or "cherry juice" sputum in our cases. 8. There is much less prostration than is the rule in pneumonia. 9. The number of cases and the time of year they have occurred are both entirely out of line with any of our previous records of pneumonia. 10. Careful bacteriologic examination has failed to show pneumococci consistently in the sputum, while rapid isolation and typing of pneumococci in cases definitely of pneumonia is our common experience.

Is acute interstitial pneumonitis a new disease? A checking of the available literature does not give us a definite answer to this question. In only four published works do we find reference to outbreaks of disease probably identical with or closely allied to this disease:

1. According to the records of the medical department of the United States army in the World War:[2]

> Certain cases of average severity, however, showed on physical examination persistent rales at either base. At Fort Sam Houston, Texas, 25 per cent of 1,000 cases of uncomplicated influenza gave this finding. There was no other evidence clinically of anything resembling pneumonia; however, x-ray plates showed local areas of increased density corresponding to the physical signs in practically all of this group. This finding is probably to be interpreted as an early or slight example of the lesion described from Walter Reed General Hospital as "hemorrhagic pneumonitis."

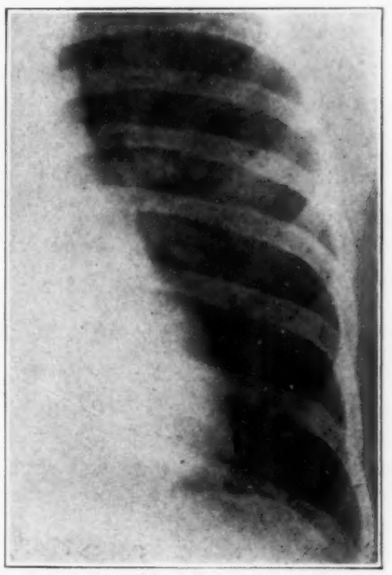

Fig. 6 (case 4).—Diffuse mottling with irregular blotchy areas of lightly increased density, typical of the severe form, on the eleventh day of illness. This may be present for two or three weeks before it begins to recede.

2. Major Albert Bowen[3] recorded an epidemic of "acute influenza pneumonitis" occurring in United States troops stationed in Hawaii from 1931 to 1934 and described the radiographic evidence as follows:

> Influenza pneumonitis involves only a portion of a lobe, usually basal, though it has been seen in the upper lobes and involving more than one lobe without increase in symptoms. It extends outward from the hilus well into the parenchyma, occasionally reaching the periphery. The roentgen appearance is

2. United States History of the World War, Washington, D. C., Government Printing Office, 1928, vol. IX, Section on Roentgenology, p. 153.

3. Bowen, Albert: Acute Influenza Pneumonitis, Am. J. Roentgenol **34**: 168-174 (Aug.) 1935.

that of a confluent mottled fan or rounded area, usually of homogeneous moderate density in the central portion, with the borders fading into the normal lung. It has the appearance of an exudative alveolar infiltration and is usually more localized and of more even density than the bronchopneumonias of childhood or than those which complicate adult diseases.

3. Dr. William H. Allen [4] reported sixty-eight cases of "acute pneumonitis" in the wards of the Station Hospital, Fort Sam Houston, Texas, in 1935. He quoted Major Albert Bowen's roentgenologic description and thus summarized the disease:

> Acute pneumonitis is used as the designation for a form of respiratory infection characterized by a benign course, few physical signs and roentgenologic evidence of a localized inflammatory process in the lung.

4. Dr. Hobart A. Reimann [1] reported eight cases of "atypical pneumonia" from the Jefferson Medical College and Hospital. He said:

> The strikingly similar clinical features of the cases reported suggest that the condition belongs to a disease group not conforming to influenza or the usual form of the common cold but included in an undifferentiated group of infections of the respiratory tract, often called tracheobronchitis, capillary bronchiolitis or bronchopneumonia.

These eight cases were similar in many respects to our few cases of serious involvement in which the pathologic process spread rather widely through both lungs. Obviously at a large city hospital it would be only the patients with rather severe or prolonged disease who would be likely to receive hospitalization, a situation quite different from that in a university or army post. In spite of this disadvantage, Dr. Reimann shrewdly deduced that his eight cases may have represented "the severe uncomplicated form of an otherwise mild and commonly encountered infection."

CONCLUSIONS

1. In a fifteen month period quite free of pandemic influenza, we have observed among the 6,569 students at Cornell University in Ithaca an epidemic of eighty-six cases of acute infection of the respiratory tract in which roentgenograms of the chest revealed typical fan-shaped areas of increased density.

2. On the evidence presented we have concluded that these areas of density were not areas of true consolidation or pneumonia.

3. We suggest that the most descriptive title of this disease is acute interstitial pneumonitis.

4. Though this is apparently a new disease in this area, it is probably identical with the "acute influenza pneumonitis" described by Major Albert Bowen as occurring in Hawaii from 1931 to 1934, with the "acute pneumonitis" described by Dr. William H. White as occurring in Texas in 1935 and with the "atypical pneumonia probably caused by a filtrable virus" described by Dr. Hobart A. Reimann as occurring in Philadelphia in 1938.

5. In areas where acute interstitial pneumonitis is found, all patients with an acute infection of the respiratory tract with a temperature rising above 101 or 102 F. should have a roentgenogram of the chest made for the purpose of excluding pneumonitis or making possible a more accurate prognosis if that disease is present.

4. Allen, William H.: Acute Pneumonitis, Ann. Int. Med. **10**: 441-446 (Oct.) 1936.

THE DETECTION OF TUBERCULOUS INFECTION

COMPARATIVE VALUE OF THE PATHOLOGIC EXAMINATION, THE TUBERCULIN TEST AND THE X-RAY EXAMINATION

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In the offices of large numbers of physicians in the United States, the examination for the presence of tuberculous infection has become as routine as that for syphilitic infection. Literally millions of such examinations are performed annually. The procedure now in practice has been extremely effective in leading to the detection of unsuspected pulmonary tuberculosis.

The Surgeon General of the United States Public Health Service, Dr. Thomas Parran, has seen the great value of this program and has said that the entire population of the United States should be tuberculin tested and that x-ray examination should be made of the chests of the reactors together with the other phases of the examination necessary to diagnose the existing tuberculosis in this country. Yet Lumsden, Medical Director, United States Public Health Service, has cast disfavor on the scheme following a small personal survey. This dissenting voice may have created doubt in the minds of a few who have not had experience with the test or are only beginning to use it.

As it is only the person who is infected with tubercle bacilli who becomes ill from tuberculosis, the first point to be determined in any medical examination is whether such infection exists. Three methods have been used to determine the presence of tuberculous infection, namely the pathologic examination, the tuberculin test and the x-ray examination.

THE PATHOLOGIC EXAMINATION

In 1876 Parrot observed that when tuberculous involvement is detected in the tracheobronchial lymph nodes there is nearly always a primary focus in the corresponding lung. In 145 cases examined he did not fail to find the lung focus. His student Hervouet emphasized the fact that the postmortem examination must be done with great care and that the making of a few sections of the lung would not suffice because some of the primary foci are extremely small, scarcely exceeding the size of a pin's head. Hutinel reported exceptions, that is, tuberculous foci in the tracheobronchial lymph nodes when no focus could be found in the lung, but he believed that this finding was due to the minute size of the pulmonary focus.

In 1898 Kuss said that primary tuberculosis in childhood cannot be diagnosed clinically but can be found at postmortem by painstaking examination. He pointed out that this condition is characterized by a small tubercle usually located just beneath the pleura, which nearly always results in involvement of the lymph nodes at the lung root. He found that the size of the focus in the lung varies from that of a hazel-nut to that of the head of a pin.

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Prepared with the aid of a grant from the Research Fund of the University of Minnesota.
From the Departments of Preventive Medicine and Internal Medicine, University of Minnesota Medical School, and the Lymanhurst Health Center.

In 1900 Naegeli found gross or microscopic evidence that tuberculous infection was present in 98 per cent of adult bodies which were subjected to postmortem examination.

In 1916 Ghon dissected in detail the organs of the chest as well as all the other organs and lymph nodes of 184 bodies. "Only in this way," he states, "was I able to detect slight tuberculous lesions in some cases where there had been positive tuberculin reaction." Because of their small size the detection of some of the pulmonary foci was "a very difficult task and often required considerable time." Again he says "The smallest foci were scarcely found at all in the cases with anatomical healing. This fact is of importance: it explains to us why it is often difficult to find such foci. . . ." In five of his cases the foci were located in organs outside the chest.

In 1933 Robertson published a report of his extensive pathologic examinations, which dealt largely with the lesions of the primary tuberculosis complex. He says: "To assert positively that any lesion during life or after death is or is not of tuberculous origin often reveals in the final analysis definite and occasionally humiliating errors. This is no expert, no matter what experience or special training he may have had, who has not been 'fooled' by this disease. . . . It is diagnosed as present when it is not. . . . The patient is assured that tuberculosis is not present when it is. Even macroscopic examination of excised tissues may fail to reveal its hidden presence. Orth demonstrated this fact six years before Koch announced the discovery of the bacillus of tuberculosis, when he found, by the microscope, lesions in grossly normal lymph nodes of animals fed on fodder infected with tuberculous material. The patient or his physician is told that the tuberculosis which he once had has been completely healed. Such a statement can never be made with any great degree of certainty and it is my purpose here to emphasize this fact."

Calcified lymph nodes and primary pulmonary foci have been ground and inoculated into guinea pigs by many workers to determine the presence or absence of virulent tubercle bacilli. Lubarsch, Rabinowitsch and Wegelin have recovered tubercle bacilli from as high as 50 per cent of such lesions. Opie and Aronson obtained positive results in 30 per cent of 169 bodies in which the lesions were thought to be apparently healed. Feldman examined microscopically and by guinea pig inoculation lesions of the primary complex from sixty-eight human beings. Although there was evidence of histologic activity in a considerable number, in only one was he able to demonstrate the presence of viable or virulent tubercle bacilli. He questions whether or not the evidence of histologic activity may not have been due to the presence of silica, which was demonstrated in these lesions. He finds that even morphologic studies cannot determine the presence or absence of viable or virulent tubercle bacilli in the lesions of the primary complex. Thus, apparently the infection completely dies out in a considerable number of the lesions of primary complexes and in others the bacilli remain alive and serve as a potential threat to the future health of the individual.

Sweany has established that pure bone develops around the foci in approximately 25 per cent of cases in man and that when the lesions of the primary complex are well calcified or ossified the capsule may be resorbed.

Thus the detailed pathologic examination is a highly accurate method of detecting the presence of the primary tuberculosis complex (tuberculous infection). Unfortunately the majority of them are located so remote from the surface that they are not available for antemortem examination.

THE TUBERCULIN TEST

Tuberculin prepared by different laboratories or even separate lots by the same laboratory may vary considerably in potency. Therefore extensive studies have been conducted by such workers as Dorset, Long and Seibert, who have determined that it is the protein fraction of the tubercle bacillus which sensitizes the tissues of the infected individual. Moreover, they have produced purified protein derivative. While this has not rendered old tuberculin obsolete—extremely valuable when prepared in such laboratories as that at Saranac Lake—it is a definite step forward. The intensive studies now in progress by Long and Seibert promise a standardized testing substance with a dosage which will be as effective in determining sensitivity of the tissues to tuberculoprotein as is possible to attain.

Lumsden says "Skin testing with any of the tuberculin preparations now on the market or otherwise amply available as a sole means of establishing an epidemiological index or rate of incidence of tuberculous infection in the general population of a community or region is of questionable value or definitely futile." I see no justification for this statement; a discrepancy (not as great as that reported by Lumsden) has been recognized for a long time. As early as 1893, Bang of Denmark called attention to the fact that in cattle the occasional animal which did not react to the tuberculin test had unmistakable tuberculous lesions demonstrated at postmortem examination. He said: "But the imperfections of tuberculin mentioned here are eclipsed by its good qualities. According to my experience, which is founded on a very great number of autopsies, it is in 10 per cent of the cases at most that tuberculin will be at fault and it is only in a very small percentage of these cases that the fault will be of consequence." In 1898 Reynolds said: "I do not mean to convey the impression that I consider tuberculin absolutely infallible, but I do consider it more nearly infallible than the human eye or sense of touch."

Every one knows that the Wassermann and other equally good tests are not infallible for the detection of syphilitic infection, yet no informed physician would condemn them and even call them futile because of the occasional failure.

There are certain factors absolutely necessary for the successful administration of the tuberculin test:

1. A high grade tuberculin with recently tested potency must be used.

2. For the individual who has not reacted to 0.01 or 0.1 mg. as the first dose, a second dose of 1 mg. should be administered in from forty-eight to seventy-two hours. When purified protein derivative is used, one should always employ the usual second dose for those who do not react to the first. The administration of a sufficient dosage of tuberculin is important, as a 10 to 20 per cent error may result from using only the small dose. For persons beyond middle life, larger final doses may be necessary.

3. If tuberculin is not lodged in the layers of the skin, no reaction will appear even though the tissues are sensitized to tuberculoprotein.

4. Interpretation is best made by palpation. In the absence of induration or edema no reaction should be recorded. Many one or two plus reactions have been based on areas of pinkness or redness and are not an indication of sensitivity to tuberculoprotein.

When adequate care is used in carrying out these four points the number of persons one misses who have foci of living tubercle bacilli in their bodies will be insignificant. If such care is not taken, of course, the results will not tally with previous records.

The evidence of the specificity of a good preparation of tuberculin properly administered now makes a voluminous literature:

1. One can administer the tuberculin test to large numbers of guinea pigs and find no reactors, then inoculate them with living tubercle bacilli; a few weeks later all will be reactors if the test is again administered.

2. Independently the veterinarians have conducted their investigation on tuberculin. The critics and advocates of tuberculin now active in the medical profession had their parallels in this profession forty years ago. The veterinarians have long since settled their controversy because the immediate postmortem examination of the carcass was proof positive of the specificity of the test. In the United States, tuberculosis among the 58,000,000 cattle, as manifested by the tuberculin reaction, has been reduced since 1917 to one half of 1 per cent or less in all but nine of the 3,134 counties. In 1917 there were 40,746 cattle condemned for tuberculosis under federal inspection in the United States; in 1938 there were only 2,385 cattle condemned. Was this futile? The veterinarian used the tuberculin test as the "sole criterion" of tuberculous infection.

3. A strain of tubercle bacillus with markedly attenuated virulence, known as BCG, when properly administered to man and animals is capable of sensitizing the tissues to tuberculoprotein.

4. We have observed 643 students of nursing and medicine who were nonreactors to tuberculin but who soon after exposure to patients suffering from contagious tuberculosis presented strong reactions. Boynton found that the rate of tuberculous infection among students of nursing in a general hospital service was 100 times greater than among students in the college of education, while among students in a special tuberculosis service it was 500 times greater than in the college of education. Among eighty-five students of nursing and medicine who developed lesions which could be located during life by one or more phases of the examination, not one failed to react to tuberculin.

5. When a contagious case of tuberculosis exists in a family, one finds that nearly all other members react to tuberculin. The presence of the tuberculin reaction among children in such families is so universal that the administration of the test to the children is often used in differential diagnosis. For example, a woman reported to one of our chest clinics with evidence of extensive disease in one lung. The x-ray laboratory reported advanced pulmonary tuberculosis. Tubercle bacilli could not be found in the sputum and she did not react to tuberculin. Finally her four small children were brought to the clinic but not one reacted to tuberculin. The final diagnosis was pulmonary abscess with no evidence of tuberculosis.

6. The tuberculin reaction among children often leads to the source of the infection. For example, Edwards of Cleveland administered the tuberculin test to first grade school children and examined the adult contacts of the reactors. In 1937, 163 and, in 1938, 224 previously unreported cases of pulmonary tuberculosis were brought to light in this manner. Can any one speak of a test which results in such accomplishments as "futile"?

7. Ch'iu found that of 446 children who reacted to tuberculin at the average age of 7 years, 15 per cent had fallen ill or had died of tuberculosis ten or more years later, whereas of 772 children who were nonreactors to tuberculin at the average age of 6.6 years only 1.7 per cent had fallen ill from tuberculosis when traced ten or more years later. Can any one say that a test is "futile" which selects the potential future cases of clinical tuberculosis in this manner?

THE X-RAY EXAMINATION

When the x-rays are used as the final diagnosis of tuberculosis, certain physical limitations of this method must be held in mind else the medical scientist will join the ranks of the awe struck public to whom x-rays are still largely magic. X-rays at their best are entirely dependent on the eyesight of the viewer. Some of the physical limitations are as follows:

1. The x-ray examination usually is limited to the chest. Ghon and other pathologists have called attention to cases in which the most minute examination at postmortem revealed no evidence of tuberculosis in the lung or hilus and yet definite foci were found in other parts of the body. Sweany has shown that about 12 per cent of the tuberculous infections are extrapulmonary. This fact immediately gives the x-rays a 12 per cent handicap.

2. On the usual single film exposed in the posterior-anterior diameter, a considerable part of the lung is obscured from view by shadows of the heart, diaphragm, and so on. Sweany has found that approximately 31 per cent of the lesions which can be detected by postmortem examination had their shadows so obscured from view on the antemortem x-ray films. Thus, an additional 31 per cent handicap must be given the x-rays in detecting tuberculous infection.

3. The x-ray examination is such a gross method that areas of disease must be macroscopic in size before they cast shadows on the x-ray film that are visible to the naked eye.

Miller's x-ray and postmortem studies of 400 cases led to the following conclusions: "With a technic of examination based upon careful study, calcification of tuberculous first infection in the thorax is demonstrable roentgenographically in only about one fourth of the existing cases. The superimposition of structures of varying density, the factors of cardiac and intrapulmonary vascular movement and the minute size of many of the calcium deposits are responsible for this. Blood vessels roentgenographed axially produce shadows which may be confused with those of calcium." Since 75 per cent of the lesions which contain calcium are not demonstrated by x-ray examination, this method cannot be set up as the standard method of detecting tuberculous infection.

Even in the small percentage of primary lesions which are located by x-ray examination it usually takes from one to several years before sufficient calcium is deposited so that it can be detected by x-ray examination. Pinner states that he has observed noncalcified primary tuberculosis complexes that were several years old and one such case in which bacteriologic evidence

of tuberculosis extended over seven years preceding death. Thus, if one were to use the x-rays to determine the presence of infection to find its source, the persons responsible for the infection would continue to disseminate bacilli or even die before their disease would be detected. On the other hand, the tuberculin reaction is definitely present from three to seven weeks after infection occurs and one can immediately seek the open case responsible for the infection. Sweany has shown that 12 per cent of the lesions found at postmortem cannot be detected by x-ray examination because they are too small or too new to cast shadows. Thus another 12 per cent handicap accrues to the x-ray examination.

4. The careful pathologic studies of Sweany have shown that many lesions of the primary tuberculosis complex become almost completely resorbed after fifteen to twenty years, thus casting no shadow on the x-ray film. There is another very definite group of primary lesions which do not form calcium deposits. This, together with those which resorb, constitutes another 25 per cent handicap for the x-rays.

Thus, according to the postmortem work of Sweany, the x-ray examination has a total handicap of 80 per cent in the detection of the lesions of primary tuberculous infection.

If the tuberculin reaction developed in only 20 to 25 per cent of animals inoculated; if it had led to the discovery of only 20 to 25 per cent of the tuberculous cattle of this nation; if it had aided us in finding only 20 to 25 per cent of infected children, certainly we would not be using it as our best standard in the detection of tuberculous infection; yet a 20 to 25 per cent effective standard has been relied on in the report of Lumsden.

5. Even the shadows we do see on the x-ray film are not diagnostic of etiology; those cast by tuberculous lesions may be identical with those of lesions of entirely different etiology. Gloyne of London says: "Thus in the case of hilus shadows an accurate phraseology was certainly needed for what we saw, but when we added the word tuberculosis, we made an inference from the observed facts which we now know to have been often wrong. A similar tendency is creeping in with regard to miliary shadows. We are apt to describe them as miliary tuberculosis. But there is a group of cases in which these shadows disappear after some months and are unaccompanied by the usual symptoms and signs of serious illness. Now miliary tuberculosis is a pathological term with a well defined meaning denoting a fatal disease and I cannot help feeling that our minds will be all the clearer if we can give to the new science of radiology a terminology of its own which cannot be confused with pathology."

Moreover, it is impossible from a shadow on the x-ray film to differentiate between shadows cast by bone, calcium or dense deposits of fibrous tissue. There are causes of calcification in the lungs and hilar region other than tuberculosis.

Frimann-Dahl and Waaler made postmortem examinations of 200 bodies which had previously had x-ray examination. They identified the primary complex in forty-two. They also studied the nontuberculous lesions which had produced shadows so easily misinterpreted for parts of primary complexes. In forty-eight cases there were ossifications and calcifications and thickened pleura not due to tuberculosis. Pieces of wood and cellulose around which calcium had been deposited were found immediately subjacent to the pleura in two cases. Ossifications between the alveoli were found in seven. Emboli and thrombi which had calcified were found in six cases. They also found the cause of the densities on the x-ray film in some cases to be due to silicotic nodules. I have seen pieces of lead in the chest and small remains of iodized oil in the bronchial tree cast shadows which were interpreted as representing lesions of the primary complex until the history revealed the true condition.

6. Krause called attention to the gradual loss of sensitiveness to tuberculin if reinfections do not occur. On a number of occasions attention has been called to the disappearance of allergy as revealed by the tuberculin test among children at Lymanhurst. This has also been observed by those working with BCG; that is, after this substance has been administered, sensitiveness of the tissues reaches its height, following which it may wane and finally disappear as far as can be determined by the tuberculin test. It is believed in such cases that the BCG has completely died out.

When all tubercle bacilli in the lesions of the primary complex have died there remains nothing except the "shells" in which they formerly lived. Tuberculosis is no longer present, regardless of the shadows that may be seen on the x-ray film. Such persons if not reinfected apparently lose their sensitiveness to tuberculin and are therefore nonreactors.

The x-rays are limited by their very nature in two opposite ways. They fail to record or at least we fail to see shadows of all the lesions whether due to minute size or obstructions in the chest and at the same time they record shadows of nontuberculous lesions as well as those of previous tuberculous lesions which no longer harbor virulent bacilli and which have no more clinical significance than an appendicitis scar.

As long as one can submit the same x-ray film to four different roentgenologists without one knowing of the readings of the others and receive three or four entirely different interpretations and as long as one can submit a film to a roentgenologist and receive a definite interpretation on one day and a month later submit the same film to the same roentgenologist and receive an entirely different interpretation, it seems more than likely that many of the differences which have been reported with reference to calcifications are due to errors in interpretation rather than to actual differences in the pathologic condition. Assuming that films have been made by the best possible technic there remains one important essential, namely that shadows be described by a physician who knows the limitations of the x-ray examination.

RECENT REPORTS OF DISCREPANCIES BETWEEN THE TUBERCULIN TEST AND THE X-RAY EXAMINATION

Recently several reports of discrepancy between the tuberculin test and the x-ray film have been made. For example, Nelson and his associates found among 198 children who reacted to tuberculin that eighty-five (42.9 per cent) had what they considered definite roentgenologic evidence of calcification, forty-three (21.7 per cent) had questionable evidence of calcification and seventy (35.4 per cent) had no evidence of calcification. Among sixty-two children who were nonreactors to tuberculin they found what they considered definite roentgenologic evidence of calcification in twenty-seven (43.5 per cent), questionable evidence in five (8.1 per cent) and no evidence in thirty (48.4 per cent). They conclude that low grade or healed tuberculous lesions

in children may exist when the tuberculin reaction is negative or only questionably positive.

Among 1,220 children examined, Crimm and Strayer state that 619 (50.7 per cent) had primary tuberculosis as evidenced by calcification in the lungs, while among 1,998 adults so examined 447 (22.4 per cent) showed evidence of such calcification. The result of the tuberculin test is not given.

Crimm and Short reported 191 cases of children and young adults who did not react to tuberculin but who they believe had at some previous time acquired the primary infection of tuberculosis from x-ray evidence of focal areas of calcification. In seventy-five persons who two or three years before were reported as one plus or two plus reactors, they found that 18 per cent had become nonreactors. Over a period of four years they saw four patients who were previously nonreactors who became reactors.

TABLE 1.—*Nonreactors to Tuberculin, January 1922 Through May 1931*

	Number
Incomplete or unsatisfactory examinations	394
X-ray examination negative	3,483
Pleural changes	91
Bronchiectasis	34
Questionable or slight calcification	76
Slight calcification	23
Moderate calcification	9
Calcified nodules throughout both lungs	2
Fibroid and calcified deposits in apex only	1
Advanced pulmonary tuberculosis	2
Pulmonary abscess	1
Pneumonia	32
Total	4,148

Gass and his co-workers examined 1,015 white and 276 colored children from 6 to 19 years of age. They submitted the x-ray films of the chests to five different observers, among whom there was unanimous agreement "as to the presence of definite tuberculous lesions in 32.8 per cent of the tuberculin-positive reactors and 24.7 per cent of the tuberculin-negative reactors. Such lesions were noted on at least four of the five observations in 39.4 per cent of the tuberculin-positive children and 46.2 per cent of the tuberculin-negative reactors."

McKneely made an x-ray survey of 6,000 children at Hagerstown, Md., and reported that 963 showed evidence of pulmonary tuberculous infection. Among 2,837 children who were nonreactors to tuberculin approximately 13 per cent had what he considered x-ray evidence, whereas among approximately 700 children who reacted to tuberculin there was x-ray evidence in 20.6 per cent. McKneely says: "In summary, tuberculin testing followed by roentgenographic examination of the reactors in the Hagerstown school children would have missed 85 per cent of the children with tuberculous lesions which were revealed by an x-ray survey. These results seem to show also that the reaction to the tuberculin test is almost unrelated to the roentgenographic diagnosis in this group of school children. Clearly, this finding appears incompatible with much that is believed to be true about tuberculosis and the tuberculin test. The first question which may be raised concerns the accuracy and correctness of our technics, and interpretations. These have been checked as carefully as possible and to date no basic error has been found." One wonders whether man's inability to determine etiology from the x-ray film is not basic enough as far as error is concerned.

Lumsden and his co-workers examined 4,400 school children from 6 to 19 years of age in Coffee County, Ala., and Giles County, Tenn. He states that in Giles County 43.2 per cent of the white school children and 25.8 per cent of the colored school children and in Coffee County only 0.6 per cent of the white school children and only 1.1 per cent of the colored school children showed lesions "regarded by the authorities generally as unquestionably healed or arrested primary lesions of tuberculosis." Among the white children examined in Giles County 46.2 per cent of the tuberculin reactors and 42.7 per cent of the nonreactors were reported to have evidence of calcium deposits revealed on their x-ray films.

If one is working in a community where approximately 100 per cent of the children have become infected with tubercle bacilli, one might expect to find about 20 per cent with evidence of the primary complexes on the x-ray film. In addition one would find similar shadows of lesions with entirely different etiology. It is possible that in some areas of this country the nontuberculous causes of densities, such as fungous disease and silicotic nodules, are more prevalent than in others. Therefore in communities where nearly all the children are infected with tubercle bacilli and the other causes of densities on the x-ray film are prevalent a much higher percentage of shadows so often interpreted as representing the primary complex will be found than in communities where the incidence of tuberculous infection among children is low and the other causes of densities on x-ray film are rare.

Among adults there appears to be a definitely lower percentage who have shadows interpreted as calcium deposits than among children. For example, an examination of 846 students by Scheel of Oslo revealed shadows without calcification which he believed were due to deposition of fibrous tissue in 6.9 per cent of the tuberculin reactors and in only 1.6 per cent of the nonreactors. He found evidence of calcified foci in from 12.2 to 16.2 per cent of the tuberculin reactors and in 0.7 per cent of the nonreactors.

Lees examined 464 medical students at the University of Pennsylvania; 338 reacted to tuberculin and thirty-nine (11.2 per cent) showed evidence of calcium deposits on the x-ray film. Of the 126 nonreactors ten (7.9 per cent) gave evidence of calcium deposits.

Among 4,372 students who entered the University of Minnesota in the fall of 1936, 1,004 reacted to the tuberculin test. An x-ray film examination was made of 982 of these, of whom only 140 (14.2 per cent) showed possible evidence of primary complexes such as calcium deposits.

Ylvisaker and his co-workers made x-ray films of the chests of 9,301 adult employees in the home office of the Prudential Insurance Company and found that 762 (8.2 per cent) gave evidence of calcium deposits in the chest.

It is possible that the reporting of definitely smaller numbers of adults than children with evidence of calcium deposits may be due in part to resorption of the calcium, bone, and so on, which Sweany has described.

OBSERVATIONS AT LYMANHURST

At the Lymanhurst School in Minneapolis we have examined 16,824 children since 1921, many of whom have been reexamined periodically. First examinations have been made of infants of a few months and of all other ages throughout the period of childhood. We have observed the same infants and children periodically from the time of the first examination as they grew into

adulthood and have seen some of them reach the fourth decade of life. Thus we have seen the complete evolution of tuberculosis in the human body from the time the individual was exposed, following which parts of the primary tuberculosis complex were occasionally detected by x-ray film in the lung or in the hilus or in both. Among such persons we have seen the primary complex make its appearance as well as acute reinfection forms of disease, such as meningitis, pneumonia, miliary tuberculosis, pleurisy with effusion, and peritonitis. We have seen chronic reinfection forms develop, such as those involving the lungs, the kidneys, numerous bones and joints and the digestive tract.

When these observations began, it was generally assumed that all children had been infected with tubercle bacilli and that therefore the x-ray examination should bring to light evidence of calcium deposits, particularly in the hilar region in the majority. For example, in 1925 in one group of 1,412 children we reported that our roentgenologists had found evidence of calcium deposits in 1,024 (72.6 per cent) and that 55 per cent of those with evidence of calcium on the x-ray film did not react to the tuberculin test. At that time the work of several authors emphasized the fact that deposits in the lung or hilus interpreted as calcium are not always due to tuberculosis.

In 1927 we reported the examinations of 4,500 children of whom the roentgenologist had described evidence of calcium deposits in 3,155 (70.1 per cent). In these 3,155 the tuberculin reaction was present in 1,683 (53.3 per cent). The same year we reported the examinations of 1,772 children among whom the roentgenologist had reported the evidence of calcification in 1,077 (60.7 per cent). Of this number said to have calcium deposits demonstrated by x-ray film, 56 per cent reacted to the tuberculin test. The conclusion was that the discrepancies between the tuberculin reaction and

TABLE 2.—*Tuberculin Reactors, January 1922 Through May 1931*

	Number
Changed from nonreactors to reactors	189
Changed from reactors to nonreactors	68
No x-ray examination	134
X-ray examination negative	2,995
Pleural changes (no evidence of calcification)	124
Pleural changes with evidence of calcification	49
Bronchiectasis, no calcium	15
Bronchiectasis with calcium	3
Questionable calcification	233
Definite calcification	765
Calcifications in cervical or abdominal regions only	5
Hodgkin's disease	1
Pneumonia (no calcium)	13
Fine fibroid deposits or nodular infiltrations in apex	16
Primary foci in pneumonic stage	41
Clinical pulmonary tuberculosis (no calcium)	24
Tuberculosis of bones and joints (no calcium)	15
Clinical pulmonary tuberculosis (with calcium)	37
Tuberculosis of bones and joints (with calcium)	10
Total	4,737

the x-ray examination "may be accounted for either on the ground that the calcification is due to some healed inflammatory condition of a nontuberculous nature, to tuberculous involvement which has completely died out or to error in interpretation of shadows." When a child was reported to have evidence of calcium deposits revealed by the x-ray film in the parenchyma or hilus, slight rotation of the chest under the fluoroscopic screen examination often would obliterate the shadow. Again, on one film of a child's chest the roentgenologist would report evidence of a definite calcium deposit in the hilar region, whereas, on the next examination of the same child's chest no such evidence would be reproted. Structures such as blood vessels seen on end were responsible for many of the reports of the presence of calcium in our previous examinations. About this time reports were

TABLE 3.—*Examinations from June 1, 1931, Through Dec. 31, 1938*

No test	12	
Total number examined	7,939	
Nonreactors	6,156	
Reactors	1,771	(22.3 per cent)
NONREACTORS		
No x-ray examination	4,987	
X-ray examination	1,169	
No abnormalities	1,064	
Possible calcium	74	
Definite evidence of calcium	18	(1.5 per cent)
Clinical pulmonary tuberculosis	2	
Pneumonia	11	
REACTORS		
X-ray examination	1,771	
No abnormalities	703	
Possible calcium	468	
Definite evidence of calcium	527	(29.7 per cent)
Primary foci in pneumonic stage	25	
Clinical pulmonary tuberculosis	34	
Pneumonia	14	

appearing in the literature calling attention to the work of others who had observed this error, notably that of McPhedran.

By 1930 the interpretations of our x-ray films were so much in error that they were really of little value in the continuation of our studies. Therefore they were reinterpreted in the light of new information.

In table 1 are grouped those who did not react to the tuberculin test and those with incomplete examination with reference to tuberculin and x-ray examination who registered in our clinic before June 1, 1931. The total group consists of 4,148 individuals, 394 of whom were not adequately examined. Thus the group with satisfactory examinations consists of 3,754 children; 3,643 (97 per cent) had no evidence of calcium deposits reported on the x-ray film, while 111 (3 per cent) had such evidence reported, although some of it was questionable. One child had definite pulmonary abscess; two had unmistakable advanced pulmonary tuberculosis. In both of them only the epidermal test was administered. It is probable that in one case desensitization had occurred, as the disease was in the terminal stage when the test was administered. In the second case such an explanation does not seem logical and, unfortunately, larger doses of tuberculin were not administered.

Among the remaining children, thirty-two had shadows in the lung parenchyma which completely disappeared on subsequent examinations. This shows the great advantage of having an opportunity to make periodic examinations. I have seen a number of such cases which on a single examination were designated as tuberculous on the basis of the x-ray shadow. No person, whether a reactor or a nonreactor to tuberculin, should ever be given a diagnosis of pulmonary tuberculosis from the shadows revealed by one x-ray examination. In not a single case of this entire group did we find a shadow in the lung which proved to represent the primary focus in the pneumonic stage.

Table 2 includes 4,737 children who reacted to tuberculin, as they registered in our clinic before June 1, 1931. However, in 134 the x-ray examination was not

done or was unsatisfactory. Of the 4,346 who had x-ray film examinations, 869 (20 per cent) were reported to have definite evidence of calcium deposits. Of the remainder there were 233 who had questionable evidence of calcium deposits on the x-ray film. Among this group of tuberculin reactors it will be observed from table 2 that twenty-five had clinical tuberculosis of the bones and joints and sixty-one had clinical pulmonary tuberculosis.

Because so little evidence of disease was found among our nonreactors, we have not made x-ray films of the chests of nonreactors since June 1931 unless they were demanded by the family or one or more symptoms aroused our suspicion. It will be observed (table 3) that only twelve of the 7,939 children who were examined between June 1, 1931, and Dec. 31, 1938, were not tested with tuberculin. Of those tested only 22 per cent reacted, while among those examined before June 1, 1931, 54 per cent reacted. Among the nonreactors (table 3) only 1,169 had x-ray film examinations and of these only eighteen presented definite evidence of calcium deposits. We have given no consideration whatever to those reported as having possible or probable calcium deposits demonstrated on the x-ray film except to list them. Clinical pulmonary tuberculosis was found in two of these nonreactors.

Among the 1,771 reactors (table 3), definite evidence of calcium deposits was reported in 527 (29.75 per cent), primary foci in the pneumonic stage in twenty-five, clinical pulmonary tuberculosis in thirty-four and nontuberculous pneumonia in fourteen.

As in the group examined prior to 1931 clinical pulmonary tuberculosis was found in sixty-one and since 1931 in thirty-four cases, making a total of ninety-five, all of whom were tuberculin reactors, and since nearly all of this group were in the teen age period, it is almost fruitless to make x-ray films of the chests of children even though they react strongly to tuberculin, before they reach the age of adolescence. It is nearly fruitless to make x-ray films of the chests of children who react to tuberculin to find evidence of the lesions of the primary complex. Therefore our studies have led us to look on the x-ray film of the chests of children as almost a total waste as far as tuberculosis is concerned.

However, when the period of adolescence is reached and on through the span of life, every tuberculin reactor should have an x-ray examination of the chest annually as long as no significant shadows appear and much more frequently if shadows appear which may be due to clinical tuberculosis. Those who present such shadows are subjected to complete examination, including laboratory studies of the sputum and the gastric contents for acid-fast bacilli, periodic red cell sedimentation tests, observation for symptoms, examination for abnormal physical signs, and frequent x-ray examinations of the chest to determine whether the shadow persists and if so whether it changes in size. Even bronchoscopic examination may be resorted to in arriving at the final diagnosis. Thus the diagnosis of tuberculosis often is made only with extreme difficulty; there are no short cuts, such as a single x-ray examination of the chest. Indeed, any one who makes diagnoses without utilizing all of these procedures is open to criticism by experienced physicians. Our experience at the Lymanhurst Health Center has been such that the term "x-ray diagnosis" has become obsolete. We have seen so many errors made that we now accept nothing except a complete examination of which the x-ray film is only a part.

THE RELATION BETWEEN VITAMIN A AND DARK ADAPTATION

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I. PURPOSE

Night blindness and similar visual disturbances have long been associated with the nutritional state of the body. In recent years this connection has been traced to the vitamin A of the diet [1] and has become understandable in terms of the chemical relation between vitamin A and the light-sensitive pigments of the retina.[2]

After the establishment of vitamin A as a factor in the visual cycle, it seemed logical to use the properties of vision for the detection of early stages in vitamin A deprivation. Such tests were made by Edmund and Clemmesen [3] with visual intensity discrimination as an index and by Jeans and Zentmire [4] with dark adaptation as a criterion. Other investigations [5] followed and, though some of this work has been found to be perhaps more enthusiastic than critical,[6] it showed that the original idea is probably sound and that under properly controlled conditions some visual properties may be used in diagnosing a lack of vitamin A long before it becomes clinically evident.

The measurements to be reported in this paper were made in an effort to study the behavior of dark adaptation under controlled vitamin A conditions and to ascertain under which circumstances the properties of dark adaptation are indicative of preclinical avitaminosis A.

II. DARK ADAPTATION

Dark adaptation is a familiar phenomenon. On coming from the brightly illuminated outdoors into a dimly lighted room, one can see hardly anything at first, but, as one stays indoors, objects slowly take shape, and after fifteen or twenty minutes they appear so clear that one recalls the initial visual obscurity with astonishment. The process of achieving this good vision is called dark adaptation. It was first described by Aubert [7] in 1865 and first measured by Piper [8] in

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An account of this work was first given March 29, 1938, to the Round Table on Nutrition and Public Health sponsored by the Milbank Memorial Fund and held at the New York Academy of Medicine. It was later reported briefly at the Vitamin Round Table of the Clinical Congress of the Connecticut State Medical Society at New Haven, Conn., Sept. 21, 1938. Part of the results have been published as a preliminary note in Science (**88:** 219 [Sept. 2] 1938).

1. Fridericia, L. S., and Holm, E.: Experimental Contribution to the Study of the Relation Between Night Blindness and Malnutrition: Influence of Deficiency of Fat-Soluble A Vitamin in the Diet on the Visual Purple in the Eyes of Rats, Am. J. Physiol. **73:** 63-78 (June) 1925. Tansley, Katherine: The Regeneration of Visual Purple, Its Relation to Dark Adaptation and Night Blindness, J. Physiol. **71:** 442-458 (April) 1931.

2. Wald, George: Carotenoids and the Visual Cycle, J. Gen. Physiol. **19:** 351 (Nov.) 1935.

3. Edmund, Carsten, and Clemmesen, S.: On Deficiency of A Vitamin and Visual Dysaptation, Copenhagen, Levin & Munksgaard, 1936.

4. Jeans, P. C., and Zentmire, Zelma: A Clinical Method for Determining Moderate Degrees of Vitamin A Deficiency, J. A. M. A. **102:** 892-895 (March 24) 1934.

5. Reviewed by Jeghers, Harold: Night Blindness as a Criterion of Vitamin A Deficiency, Ann. Int. Med. **10:** 1304-1334 (March) 1937. For more recent references see Isaacs, Jung and Ivy.[6]

6. Compare especially the following critical papers:
Palmer, C. E., and Blumberg, Harold: The Use of a Dark Adaptation Technic (Biophotometer) in the Measurement of Vitamin A Deficiency in Children, Pub. Health Rep. **52:** 1403-1418 (Oct.) 1937.
Isaacs, B. L.; Jung, F. T., and Ivy, A. C.: Vitamin A Deficiency and Dark Adaptation, J. A. M. A. **111:** 777-780 (Aug. 27) 1938.
Gridgeman, N. T., and Wilkinson, H.: Night Blindness and Vitamin A Deficiency: The Use of the Biophotometer, Lancet **1:** 905 (April 16) 1938.

7. Aubert, H.: Physiologie der Netzhaut, Breslau, 1865.

8. Piper, H.: Ueber Dunkeladaptation, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **31:** 161, 1903.

1903. Since then it has been extensively studied, and now there exists a body of information which is sufficient to describe it quantitatively.[9]

Measuring dark adaptation involves finding the minimum light which must illuminate a given surface in order to render it visible. This threshold illumination is high when one enters a dark room and decreases as one stays in the dark. The most striking thing about this change in visual threshold is its range; it can easily cover a gamut from 100,000 units of light intensity at the beginning to 1 unit at the end of dark adaptation.

The retina is neither a simple nor a uniform structure. It possesses two kinds of receptors—rods for vague colorless vision at low light intensities and cones for acute color vision at high intensities—and these are distributed in a definite and characteristic pattern over the surface of the retina.[10] Since dark adaptation is accomplished by both rods and cones, their distribution and properties determine the precise nature of the dark adaptation corresponding to any region of the retina. Unless understood, these complexities will trip the innocent investigator who is unaware of the accumulated visual knowledge of the last twenty-five years.

It would obviously be out of place to present this information here, especially since it has already been reviewed in detail elsewhere;[9] therefore we may state categorically that there are at least six specifications which must be made in order that measurements of dark adaptation may be numerically precise and quantitatively valid. These conditions are (1) the intensity and (2) the duration of the light present before dark adaptation begins and (3) the area, (4) the retinal location, (5) the color and (6) the duration of the light used for measuring the course of dark adaptation.

In order to understand what these specifications involve, it is well to look at the measurements of dark adaptation shown in chart 1. For the moment consider only the upper set of points in the chart. They are single measurements which record the light intensity which the observer can just barely see as he stays in the dark. Time is plotted on the horizontal axis on an ordinary linear scale. Light intensity, however, is plotted on the vertical axis on a power or logarithmic scale. On such a scale 1 means 10 units, 2 means 100 units, 3 means 1,000 units, and so on. The most important reason for using a logarithmic scale is the large range of intensities covered by the data. A simple linear plot would have to be enormous to show any detail in such a range and would cramp some portions of the data and exaggerate others. Another virtue of a log plot is that a percentage error always occupies the same distance; a 10 per cent error is the same size whether applied to 1 unit or to 1,000 units.

Chart 1 shows that dark adapation proceeds in two steps. The first is rapid and is over in a few minutes; the second is late in starting and goes on for a half hour or more. It is now well known that the rapid first adaptation records the behavior of the cones of the retina, while the delayed and slower second adaptation reports the function of the rods. When, as for chart 1, the measurements have been made with violet light, this is brought out by the sharp and easily noted color difference between those thresholds which are determined by the cones and those made by the rods. Even a completely inexperienced observer records the fact that at the threshold the lights corresponding to the first section (solid circles in chart 1) appear violet or blue, while those composing the second section (unfilled circles) show no color at the threshold.

The six conditions under which the measurements are made influence profoundly the relative extent, the duration and even the form of the two parts of the dark adaptation curve. Thus the brightness and duration of the light adaptation which precede dark adaptation will fix the time of appearance of the cone-rod transition; after prolonged exposure to high intensities, the break may not come before fifteen or sixteen minutes, whereas after short exposure to low intensities it may come after half a minute or even less. The same is true of the color of the light used for measuring the threshold. Measured with red light, dark adaptation may show mainly the first, cone section, followed perhaps by a very slight rod section, which appears after thirty minutes; while with violet light

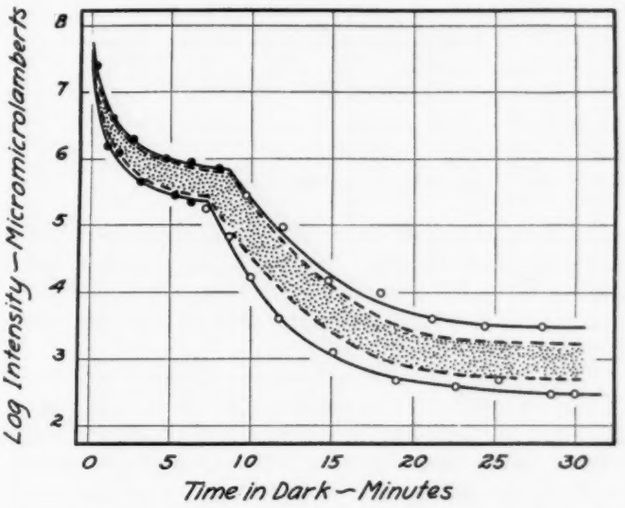

Chart 1.—The course of dark adaptation in 110 normal persons. The points represent the single measurements made with two subjects yielding the highest and lowest values of the final threshold. The dotted area contains 80 per cent of the population. Note the cone primary adaptation marked with filled-in circles, the rod secondary adaptation marked with unfilled circles and the cone-rod transition time.

the cone portion may hardly show, and only the rod, secondary section will be dominant. Similarly the area and location of the part of the retina used in the tests share with the other factors the control of the course of dark adaptation.

III. STANDARD CONDITIONS AND APPARATUS

Since all these circumstances determine the nature and course of dark adaptation, it took many preliminary tests and a long experience with visual measurements for us to formulate the best conditions for routine measurements. The conditions we finally adopted as standard were chosen for their physiologic reasonableness.

To begin with, the precise effects of nutritional and pathologic conditions on the course of dark adaptation are not known. We therefore chose such a light adaptation that both cone dark adaptation and rod dark adaptation are separately and adequately shown in the measurements. This means a light adapting field, occupying about 35 degrees visual angle, whose brightness is about 1,500 millilamberts. The exposure

9. Hecht, Selig: Rods, Cones and the Chemical Basis of Vision, Physiol. Rev. **17**: 239-290 (April) 1937.
10. Østerberg, G.: Topography of the Layer of Rods and Cones in the Human Retina, Acta Ophth., vol. 13, suppl. 6, pp. 1-103, 1935.

to it is for three minutes, which is long enough to produce the main effect and short enough to keep the subject from being bored.

The test field occupies 3 degrees visual angle in diameter as a compromise between a smaller field, which reports the behavior of a uniform part of the retina, and a larger field, which is more easily seen by untrained subjects. The test field and the light adapting field are viewed 7 degrees nasally with the right eye. This position was selected because at this point the population of retinal rods and that of cones are more nearly

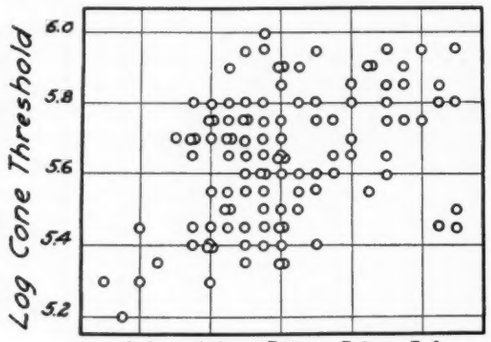

Chart 2.—Relation between final rod threshold and final cone threshold shown by 110 persons. The two thresholds are obviously connected, but the correlation is less than half perfect. As chart 4 shows, age is the biggest factor in disrupting the correlation.

equal than in the center of the retina or farther in the periphery. Moreover, the luminous fixation point used to keep the eye centered and steady is thus sufficiently far from the measuring area so as not to interfere with its function.

The measuring light is from the extreme violet end of the spectrum. This makes the rod section always maximal in range and furnishes the clear color distinction between cone function and rod function already referred to and shown in chart 1. The measuring light is exposed in flashes of one-fifth second, which is long enough to produce a good perception of the test area and short enough to be near the retinal action time.

These specifications may be duplicated by any one who will set up apparatus for controlling intensities, times, positions and areas. However, for convenience Hecht and Shlaer[11] have designed an adaptometer which not only incorporates these specifications but is so arranged that they can be varied in case it is necessary to investigate other aspects of dark adaptation under different but controlled conditions. This apparatus and the mode of its use have been described in detail and therefore need not be reproduced here. Throughout the present research we used an apparatus of this design and followed the recommended procedure.

In barest outline, this procedure consists of two parts. First the eye is exposed to the standard light adaptation. Then as the subject stays in the dark the intensity threshold of the selected region of the retina is measured with flashes of violet light. The exact steps in the measurements are important, and the various parts of the apparatus are concerned merely with carrying them out expeditiously and with precision. Each datum secured represents the just perceptible light intensity at a certain time in the dark, and between ten and fifteen such determinations yield a curve of dark adaptation similar to those shown in chart 1.

Measurements of dark adaptation made in this way reach a final threshold whose brightness is about one thousand-millionth of a lambert. This brightness represents approximately the appearance of a white card illuminated by a candle 1,000 feet away and corresponds roughly to an illumination of one millionth of a foot candle. The logarithms of such fractional numbers are negative and inconvenient to use. We therefore adopted a unit much smaller than the lambert: this is the micromicrolambert ($\mu\mu$l), which is a lambert $\times 10^{-12}$, or one million-millionth of a lambert. All possible values of the threshold are thus given by positive logarithms.

IV. NORMAL VARIATION

Before making any dietary experiments, we wished to ascertain the normal distribution of the characteristics of dark adaptation. For this purpose we made routine measurements with 110 persons, most of whom had university associations, as faculty, students, wives or children. The reason for choosing university people is not merely the obvious one of convenience but rather the practical one of normality. University people are more likely to represent a well fed group, well balanced in its dietary habits. That this judgment is right will be evident later. The measurements are all single readings and first performances. This makes them comparable to measurements which are likely to be made for ordinary diagnostic purposes.

Chart 1 gives the type of result we secured. The two sets of measurements actually shown are of the two persons who yielded the extreme final rod thresholds, the highest and the lowest. The stippled band between the two dashed curves is drawn to include the data of eighty-eight people, or 80 per cent of our population. The chances are therefore quite high that

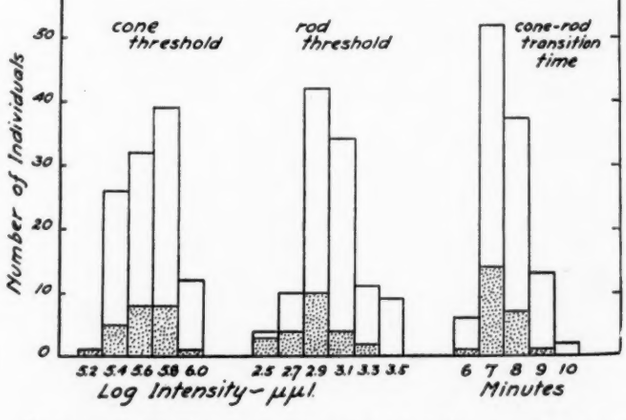

Chart 3.—Distribution of the three dark adaptation properties in the population. The stippled areas represent females. Obviously sex does not affect the distribution.

an average person when tested will show a dark adaptation curve which falls in this band.

It so happens that this stippled area in the cone region actually contains 93 per cent of all the cone measurements. This is because cone threshold and rod threshold are only partially correlated. Chart 2 records the final cone and rod thresholds of our population. It is evident that there is a general tendency

11. Hecht, Selig, and Shlaer, Simon: An Adaptometer for Measuring Human Dark Adaptation, J. Optic Soc. America **28**: 269-275 (July) 1938. This instrument was made for us by Mr. O. C. Rudolph, 55 Van Dam Street, New York City.

for the two thresholds to vary in the same direction but that the correlation is not high. Determined in the usual statistical manner the correlation coefficient of the two final thresholds is 0.44, where 1 is a perfect correlation and 0 is no correlation.

After studying the various properties of the course of dark adaptation, such as the speed of adaptation and time for the cone-rod transition, we selected three aspects which seem to possess quantitative value as description. These are the final cone threshold, the final rod threshold and the cone-rod transition time. Chart 3 shows the distribution of these properties in our population. The cone threshold distribution appears slightly skew; the other two distributions are approximately symmetrical. The stippled areas represent females and the clear areas males. Obviously sex does not affect the distribution.

Age has some influence on dark adaptation. This is most clearly evident on the final cone threshold. Chart 4 shows that the cone threshold slowly rises with age, as evidenced by the position of the distribution maximum for the different age groups. Because violet light is used in these measurements, it is possible that the rising cone threshold is due to the yellowing of the ocular media with age. In that case the same rise in the threshold should be shown by the rods. However, the rod threshold shows no such obvious change with age. There is a subtle shift in distribution of rod thresholds with age which deserves to be pointed out, but we lay no emphasis on it. Note in chart 4 that for the 15 to 20 year group the distribution is skew to the left of the maximum, for the 21 to 25 year group it is symmetrical, for the 26 to 39 year group it is definitely skew to the right of the mode and for the 40 to 65 year group it is strongly skewed to the right. On the other hand, the cone-rod transition time is unaffected by age, the distribution maintaining the same maximum and the same degree of asymmetry throughout.

How much of the spread covered by the measurements is day-to-day variation and how much is fixed individual difference? Our experience with visual measurements indicates a maximum day-to-day intensity variation of 0.3 log unit. It may well be that for several days or even weeks of measurement of a visual function a person is consistent to within less than 0.1 log unit. Then, perhaps after a short lapse of time or quite suddenly as the result of fatigue or of a night out or even for no apparent reason, he may change and give values which fall within about 0.3 of the previous readings. Previous measurements with dark adaptation follow this expectation. However, for routine purposes we chose six subjects at random and measured them five or six times in four weeks. The resulting threshold variation for each person corresponds with previous experience, and it would serve no useful purpose to reproduce the measurements. A range of 0.3 log unit is a factor of 2 in actual light intensity. This means that, if the threshold today is 1, next week it may be 2 and it will continue to lie between these values. Considering that the total threshold change during dark adaptation is about 4 log units, or 10,000 to 1, this is a comparatively small variation.

This day-to-day fluctuation is not due to experimental or instrumental errors. In the apparatus we use, it is a simple matter to set a reading to 0.1 log unit, and a careful investigator can set it to considerably less. The variation encountered among the population therefore represents not only the day-to-day fluctuations that any individual might be subjected to but in addition real differences between the individuals.

V. BASIS FOR INDIVIDUAL DIFFERENCES

The total spread of final rod thresholds in charts 1 and 3 is 1.0 log unit, or 1 to 10 in linear intensity units. Deducting 0.3 log unit as the expected day-to-day variation leaves 0.7 log unit as the range within which individuals vary one from another. It is naturally tempting to suppose that the distribution in this range is due to the variation of the vitamin A content of the diet and of the body and that an individual's position in the population spread depends on his normal eating habits. Our experiments do not bear this out.

Of the eleven persons whose final rod thresholds lie in the extreme upper range, we selected six for study. Their diets seemed well balanced and contained plenty of vitamin A, and none of them showed any of the usual clinical signs of A deficiency; moreover, none of

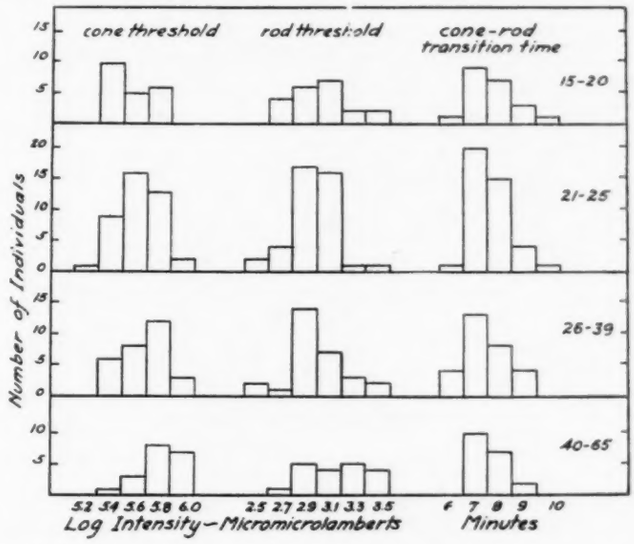

Chart 4.—Distribution of dark adaptation characteristics according to age. The cone threshold is most affected by age, the rod threshold only slightly and the cone-rod transition time not at all.

them had noticed any subjective difficulties in dark adaptation. We fed them 50,000 units of vitamin A daily for three and four weeks in addition to their regular diet. Five of the six showed no changes in their dark adaptation curves during this period, that is no change that did not fall within the normal day-to-day variation evidenced by any person whose curves are measured repeatedly. Obviously neither the daily fluctuations nor the differences between normal persons can be attributed to the daily fluctuations or the consistent differences in the vitamin A content of the diet. This is also confirmed by the case of one of the investigators in our laboratory whose final rod threshold has remained consistently among the highest despite the fact that he has eaten all sorts of supplementary vitamin A and carotene in the last two years.

Our study is thus in contrast to the investigation of 162 medical students in Boston made by Jeghers.[12] He reported that fifty of these students were subnormal in their dark adaptation. Of the fifty, at least twenty-seven were described as clinically aware of their poor dark adaptation and fifteen as possessing cutaneous

12. Jeghers, Harold: The Degree and Prevalence of Vitamin A Deficiency in Adults, J. A. M. A. **109:** 756-761 (Sept. 4) 1937.

symptoms in addition. All those tested improved under treatment, most of them strikingly. Our own group contains sixty students, undergraduates and graduates. Their dark adaptation characteristics dominate the first two age groups in chart 3 and do not differ from those of the other groups except as age influences their distribution. Actually our six persons with high thresholds are all students, and five of them showed no change with supplementary feeding of vitamin A. The sixth will be discussed in a moment.

The differences between our subjects and those of Jeghers may be quite real; however, these differences may be exaggerated by the apparatus used by Jeghers, namely the biophotometer. Other studies with this instrument have previously failed of complete corroboration, and the instrument itself may be criticized on several grounds. In particular, Jeghers[12] reported a sharp correlation between biophotometer readings and the estimated vitamin A content of the diet of his medical students. Such a correlation has failed to appear in a similar study by Isaacs, Jung and Ivy,[6] who have shown that the statistical errors involved in

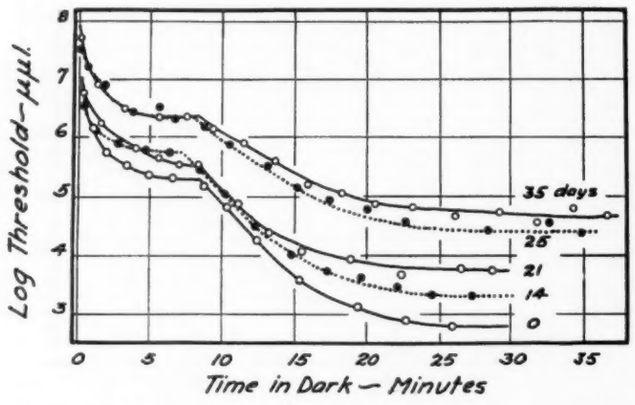

Chart 5.—Dark adaptation measurements made with one person at various times in the course of a diet practically free from vitamin A. The filled and unfilled circles merely keep the different sets of measurements apart and are smoothed by dotted and continuous lines respectively. Compare this chart with chart 1 and note how soon the data reach a level above the normal.

the use of the biophotometer are too large for such a study to be significant. The biophotometer does not incorporate in its structure and recommended procedure many of the critical specifications for measuring dark adaptation already referred to and fails to link its results with physiologic knowledge. One of the major errors is the lack of a fixation point; this failure to define the retinal area measured probably results in considerable erratic variation among individuals depending on the skill and aptitude of the subject.

In our study, even though in general the position of a normal person in the threshold spread is probably determined by other things than his diet, it is conceivable that one whose threshold is normally low may, on account of a deficient diet or of another bodily condition, be shifted to a higher position in the threshold range. Of the six subjects with high thresholds selected, one turned out to be such a person.

To begin with, his final rod threshold corresponded to log I = 3.5 and his cone-rod transition time to ten minutes, both of which are maximal in our population. Like the other five persons, he had an apparently normal diet. Yet, unlike that of the others, his dark adaptation was distinctly altered by the supplementary feeding. At the end of a week his final rod threshold had fallen from log I = 3.5 to 3.0 and at the end of three weeks it reached nearly 2.5. Moreover, during this time his cone-rod transition time had shortened steadily from ten minutes to six.

It will be apparent in a moment that ordinary dietary avitaminosis A does not change the cone-rod transition time, even though it may alter the cone and rod thresholds considerably. On the other hand, avitaminosis due to cirrhosis of the liver alters not only the thresholds but also the cone-rod transition time, as Haig, Hecht and Patek[13] showed. Apparently catarrhal jaundice also changes the cone-rod transition time.[14] We are therefore inclined to think that the extreme position of this individual in the population, as well as his exceptional response to supplementary feeding of vitamin A, is due to some aberrant factor in his vitamin A metabolism which may later become apparent clinically.

VI. DIETARY AVITAMINOSIS

After having established the spread in the dark adaptation of a normal population, we studied the dark adaptation of four young men on a regimen of decreased vitamin A. First we measured their dark adaptation almost daily for two weeks while they continued their normal and apparently adequate diet. Then we restricted their diet to foods that were calculated to yield at most 150 units of vitamin A daily. In this we followed the data compiled by Daniel and Munsell.[15] No special supplements were given of the other vitamins, because we wished to imitate the conditions under which dietary avitaminosis A would most commonly become established. The diet was maintained for about forty days, after which all four men returned to their normal food habits. Two were given daily supplements of 50,000 units of vitamin A for about six weeks afterward.

In studying the data during this experiment we followed the various properties shown by the dark adaptation curves. In particular we wanted to discover how the three selected characteristics shown in chart 3 behaved during the tests, because in the experiment with dietary avitaminosis A made by Jeghers[12] on himself the measurements were performed with inadequate apparatus and procedure and failed to show what actually happened to the course of dark adaptation. In the recent and similar experiment reported by Wald, Jeghers and Arminio,[16] made since our preliminary notices, the procedure and apparatus have been changed to render the results more revealing of the properties of dark adaptation.

Chart 5 shows five curves of dark adaptation made with one of our subjects during different stages of the deficient diet. Of the three critical properties it is apparent that both the cone thresholds and the rod thresholds are affected by vitamin A deprivation. Rod vision is affected more than cone vision, as evidenced by the more rapid and extensive rise of the rod section during the diet. This confirms the work of Haig, Hecht and Patek,[13] who found more extensive changes in rod function than in cone function in the dark adaptation of patients with cirrhosis of the liver. It is confirmed by the measurements of Wald, Jeghers and Arminio.[16]

13. Haig, Charles; Hecht, Selig, and Patek, A. J.: Vitamin A and Rod-Cone Dark Adaptation in Cirrhosis of the Liver, Science **87:** 534-536 (June 10) 1938.
14. Haig, Charles: Personal communication to the author.
15. Daniel, E. P., and Munsell, H. E.: Vitamin Content of Foods, Miscellaneous Publication 275, United States Department of Agriculture, 1937.
16. Wald, George; Jeghers, Harold, and Arminio, Joseph: An Experiment in Human Dietary Night Blindness, Am. J. Physiol. **123:** 732-746 (Sept.) 1938.

Differing from the clear effect of the diet on the cone and rod thresholds is the complete lack of effect on the cone-rod transition time; this remained the same during all the vicissitudes of the diet for all four subjects.

The parallel behavior of rod and cone functions is shown in chart 6, which records the results with the two subjects who were given supplements of vitamin A after their return to a normal diet. The data of J. M. are about average for the four subjects, while L. W. represents the extreme effects. Each point in chart 6 is the final threshold either of the cone section or of the rod section of such curves as those in chart 5. The thresholds of the last two runs on the normal diet are shown above the diagonally shaded area on the time axis of chart 6; the clear area records the duration of the deficient diet and the black area the subsequent normal diet, supplemented as already described. It is not necessary to stress the similarities of the changes in cone and rod vision. The results confirm those of Haig, Hecht and Patek for the cone and rod thresholds during vitamin A therapy of alcoholic subjects with cirrhosis and are in turn corroborated by Wald, Jeghers and Arminio's study of one instance of dietary avitaminosis.

The important thing which chart 6 brings to light is that, as measured by dark adaptation, the presence of a dietary avitaminosis becomes evident after the very first day of the absence of vitamin A in the diet. Because of the normal day-to-day variation no reliance can be put on such a single early measurement, but the rise in threshold continues steadily and in less than a week of vitamin A lack the rod and cone thresholds in chart 6 have mounted well beyond their daily fluctuations, and a conclusion of decreased visual function can be reached with reasonable certainty. Note particularly that after about ten days of the diet the rod threshold has risen beyond the commonly encountered range included in the band in chart 1 and that a few days later it has reached a value which is higher than the extreme found in only 3 per cent of our normal population. This clearly indicates the normal character of our own population, since so short a period of deficiency sends the measurements beyond the common range.

During this period of a deficiency diet our subjects were instructed to report any subjective or other symptoms noted; with one exception none were reported, though at least one subject did a fair amount of night driving. The one report was made by L. W. toward the end of the period, when he recorded that he had collided with a wall in his home and was surprised that he had not seen it. Evidently most night illumination is well above the threshold of vision.

The return to a normal diet with or without daily supplements of vitamin A did not result in the spectacular recovery of the visual function reported by Edmund and Clemmesen [3] and by Jeghers.[12] As chart 6 shows there is a slightly rapid drop in threshold at first, but this is followed by a gradual decrease in threshold, and it took about two months for our subjects to return to the normal range.

Several times after the administration of a dose of vitamin A we followed the dark adaptation of an experimental subject at intervals during the day, but we observed no striking change. Chart 6 shows one such instance, which is perhaps the only effect that we could call a positive one. It concerns the three points in the rod data of L. W. above the narrow black area. This records a single administration of 100,000 units of vitamin A, and the points above it show the cone and rod thresholds determined in the usual way before the dose and three and six hours after it. The drop in rod threshold is about 0.4 log unit, which, considering the variation shown by L. W. during the remainder of the experiment, is nothing to be certain about and is in a different category from the almost immediate drop to normal reported since by Wald, Jeghers and Arminio.[16] A possible reason for this divergence in results may be that their subject was given supplementary vitamin B during the deficient diet, while ours took only the amounts present in the restricted diet. But this is not the case with the dramatic recoveries reported by Edmund and Clemmesen.[3] Further work will be necessary to clear up this discrepancy.

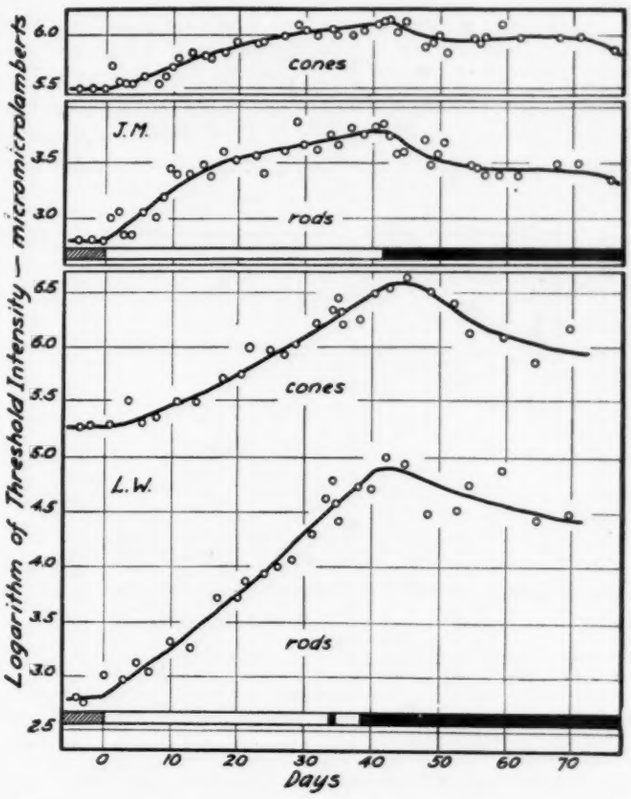

Chart 6.—The final cone and rod thresholds of two subjects on a diet nearly free from vitamin A. The diagonally shaded part represents the normal diet, the clear part the diet free from vitamin A and the black part the normal diet plus 50,000 units of vitamin A daily.

VII. DARK ADAPTATION AND DIAGNOSIS

As a result of these observations we feel certain that measurements of dark adaptation when carried out under properly standardized conditions can be used as an aid in the determination of the vitamin A condition of the body. Normal dark adaptation means a normal vitamin A content of the body, and an abnormal dark adaptation means a disturbance in the vitamin A content.

What normal is remains to be defined precisely, but this definition must be made by others. At present we may take it to mean the dark adaptation and vitamin A content of ordinarily well nourished, healthy persons such as we chose for our survey. It may well be that the common, normal dark adaptation can be improved by supplementary feeding of vitamin A. Our own attempts in this direction have not been successful, but they lasted only from three to four weeks, and more prolonged supplementation may be necessary. How-

ever, even if the commonly found dark adaptation could be improved in this way, it remains to be shown that improvement is desirable. For the present therefore normal had better be accepted in its ordinary usage.

When the dark adaptation of a person is found to be abnormal, it may mean either that his retina is not adequately supplied with vitamin A or that his visual apparatus is defective. Such defects may be congenital, like the absence of cone or rod function, or they may be acquired pathologic disturbances. When these genetic and pathologic possibilities have been excluded, one must next determine the basis for the lack of retinal supply of vitamin A. It may be the diet, in which case the remedy is simple. It may, however, be some other link in the metabolic chain from diet to retina, such as the liver, in which case other signs must be looked for.

It is these possibilities which make us doubtful of the recommendation by Wald, Jeghers and Arminio [16] that the final rod threshold alone may be adequate as a diagnostic sign. Since the final cone threshold also changes during avitaminosis—dietary and pathologic—the recognition of a change in its value serves as a corroboration of the rod data. Moreover, it is not the final threshold

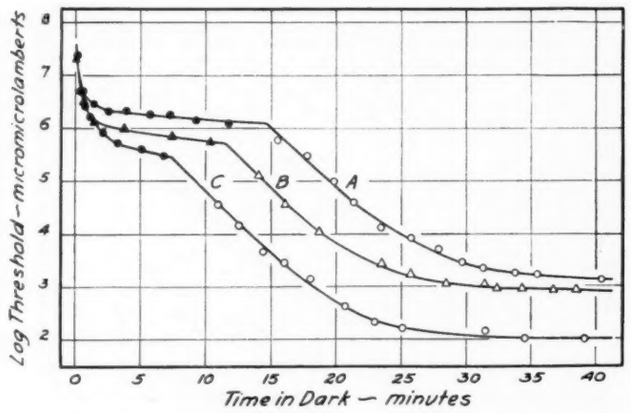

Chart 7.—The dark adaptation of a patient with cirrhosis of the liver, taken from the preliminary report of Haig, Hecht and Patek.[13] The conditions of measurement were slightly different from those here used but near enough for adequate comparison. Curve A was obtained when the subject was on an ordinarily adequate diet, while curves B and C were obtained after 105 and 127 days of intensive vitamin A therapy. Note especially the decrease in cone-rod transition time, as compared with its stationary position in the experiment with dietary avitaminosis recorded in chart 5.

alone which changes but the whole position of the dark adaptation curve on the intensity axis, and it is with much more confidence that one can conclude from the curve as a whole rather than from the position of its final point.

More significant than either of these reasons is the fact that other properties of dark adaptation are also subject to change, and it may be that further study will show characteristic disturbances of adaptation resulting from various diseases. Thus simple dietary avitaminosis does not change the time of the cone-rod transition point, whereas severe cirrhosis of the liver delays its appearance to almost twice the time, even though the final thresholds may be affected to the same extent in the two instances. We think this so important that we reproduce in chart 7 the curves from Haig, Hecht and Patek's study of dark adaptation in an instance of cirrhosis of the liver. Curve A was obtained when the subject had been on a normally adequate diet for three weeks, while curves B and C were obtained after 105 and 127 days of intensive vitamin A therapy. Compare this chart with chart 5 and note especially the way the time for the cone-rod transition gradually shifts to normal. It may well be that this difference in behavior of the cone-rod break is purely a matter of short period avitaminosis versus prolonged deprivation. This in itself would be of significance in diagnosis. For such reasons we feel that it is better to determine the dark adaptation curve as a whole rather than just the position of the final rod threshold.

These reasons apply equally well to the calls that are sure to be made for an apparatus for rapid diagnosis. This is a desirable trend, especially for extensive survey purposes, but we hope that it will not be acted on soon. A rapid procedure can consist only of a shorter light adaptation followed by the determination of the time to reach one or two fixed intensities during dark adaptation. To be reached rapidly these fixed points must be on the cone curve, so that one will lose the value of the cone-rod transition time and of the final rod threshold as aids in diagnosis. It might seem that if one started with a low enough intensity of light adaptation the resulting adaptation would be purely a rod function even if rapidly made. However, for the low light adaptation to be effective it must be preceded by a preliminary period of dark adaptation, which defeats the aim of a quick test. In short, a rapid test can measure only cone adaptation, and too little is known at present of the effects of disease and diet on dark adaptation for any one to select with confidence a point on the curve for use as a fixed criterion. We therefore urge, for a while at least, that authors performing clinical and physiologic investigations with dark adaptation and vitamin A record the whole curve of dark adaptation in their procedure.

SUMMARY

The dark adaptation of a university population of 110 persons, when measured under standardized conditions, shows three properties which are useful quantitatively: the final cone threshold, the final rod threshold and the time for the transition from cone function to rod function. The distribution of these properties is not affected by sex. Age definitely increases the cone threshold but affects the rod threshold only slightly and the cone-rod transition time not at all.

The range over which these properties vary in the population is composed partly of day-to-day variation and partly of individual difference, the latter causing much the wider spread. Individual differences were not the result of differences in the vitamin A content of the diet, and with a solitary exception no significant changes were produced by supplementary vitamin A feeding of persons with the highest thresholds.

Four young men when deprived of dietary vitamin A responded by a steady rise of both cone and rod thresholds which was apparent almost from the first day after the removal of vitamin A from the diet. After about two weeks' deprivation they showed thresholds above any values normally found in the population; their thresholds continued to rise so long as the dietary deprivation was maintained. The return to a normal diet with or without supplementary vitamin A resulted in an initial small rapid drop in threshold followed by a gradual decrease; nearly two months was required for the subjects to return to normal.

Measurements of dark adaptation when made under critically standardized conditions can be used as an aid in the diagnosis of avitaminosis A produced not only by a lack in the diet but by a functional disarrangement in the flow of vitamin A from diet to retina.

THE ENDOCRINE IMPLICATION OF JUVENILE CHONDRO-EPIPHYSITIS

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FRED L. STRICKROOT, M.D.
AND
FRANK H. PURCELL, M.D.
DETROIT

Juvenile chondro-epiphysitis is described as an alteration in shape of the developing epiphyses associated with an apparent aseptic necrosis. Pathologically there may be rarefaction or even cystlike formation and degeneration. Clinically there is possibility of eventual deformity. Roentgenographically it is characterized by the following changes in the developing osseous centers: diminution in size, decrease in density, rarefaction, fragmentation, irregularity of the ossification contour and in general a fuzzy, ragged appearance of the chondro-epiphysial structures. Depending on the various structures showing such x-ray changes, almost innumerable proper or descriptive names have been employed. This conglomeration of proper names is confusing in conditions which, we think, are identical and have the same etiology.

The suggested etiology up to the present time has been entirely theoretical without any modicum of fact. The current popular impression leads one to believe that it is the result of some inflammatory reaction or blood vessel anomaly resulting in poor vascular nourishment. It also has been thought that it is a uni-epiphysial disturbance and that clinically it is associated with pain and swelling.

The significance of this publication is an attempt to prove that juvenile chondro-epiphysitis is due to an endocrine imbalance, specifically to a primary or secondary hypothyroidism in which the tissue differentiating factor is absent, that it is a generalized process involving one or more of the developing chondro-epiphysial structures and that it is at least incipiently asymptomatic.

Engelbach some twenty years ago pointed out the value of the roentgenographic study for osseous development in clinical endocrinology. It ranks today as one of our most useful diagnostic procedures. In the younger age group, from birth to 8 years, it pertains specifically to thyroid function and is related to the time of appearance of the various centers. A delay in appearance of the centers is pathognomonic of a hypothyroid state due to the absence of this same tissue differeniating factor. Certainly in this age group the x-ray by far outranks the basal metabolic rate for accuracy because of the evident difficulty in evaluating the latter test in younger children with the ordinary clinical apparatus available.

In the older age group, from juvenility to completion of adolescence, the epiphyses are all present and in a fair state of development. In this period delayed or advanced union gives visual x-ray evidence of gonadal function, early union being indicative of premature adolescence or sex maturity, delayed union being indicative of a gonadal hypofunction.

In 1931 we began to do routine x-ray studies on all children with suspected endocrinopathies, following the plan originally devised by Engelbach and his colleagues Tierney, MacMahon and Shelton.[1] Hence not one joint but from two to four were roentgenographed, depending on the age of the patient. Very early in our studies of endocrine dwarfism we were struck by the frequency of single or multiple involvement of the chondro-epiphysial structures and so originally reported our observations.[2] It soon became apparent that this condition was not peculiar to dwarfism but was being seen with as great or greater frequency in other endocrine conditions displaying signs of hypothyroidism.

The basis for this report is the study of (1) a group of 258 clinically proved endocrinopathic patients in the age range of 8 to 15 years and (2) a control group of ninety-nine assumed normal children in the same age group. Out of the 258 endocrinopathic patients studied ninety-one, or 35.2 per cent, showed a chondro-epiphysial disturbance. Two hundred and eighteen of this group displayed clinical evidence of primary or secondary hypothyroidism, such as roentgenographic evidence of delay in osseous development, decreased basal metabolic rates, increased blood cholesterol or developmental delay. The number of hypothyroid cases showing chondro-epiphysial disturbance was eighty-five, or 39.0 per cent; in other words, all but six cases of chondro-epiphysitis were associated with hypothyroidism. Eight hundred and ninety-four joints were roentgenographed, an average of 3.46 roentgenograms per patient. It is believed that the incidence would have been materially increased if more or if all joints had been roentgenographed as a routine. Thirty-eight, or 41.7 per cent, of the ninety-one showing involvement showed multiple epiphysial disturbance. An unusual observation in table 1 is the most frequent involvement of the trochlea of the humerus. More than casual survey of the literature failed to reveal chondro-epiphysitis

TABLE 1.—*Site of Occurrence of Osteochondritis*

Site	Number of Cases in This Series	Original Reporter
Trochlea of the humerus	44	
Epiphysis of os calcis	40	Haglund, 1907
Tibial tubercle	26	Osgood, 1903; Schlatter, 1903
Olecranon	11	
Greater trochanter of femur	8	
Lesser trochanter of femur	..	Monde Felix, 1922
Head of femur	5	Legg, Calvé, Perthes, 1910
External condyle of humerus	4	
Capitellum of humerus	3	Panner, 1927
Patella	3	Köhler, 1908
Tip of acromial process	1	
Distal epiphysis of tibia	1	Stein, 1922
Head of radius	1	
Distal epiphysis of radius	1	
Distal epiphysis of ulna	1	Burns, 1931
Tarsal scaphoid	1	Köhler, 1908
Head of humerus	1	Hass, 1921
Greater tuberosity of humerus	1	Hass, 1921; Lewis, 1928
Heads of metacarpals	..	Maneiarie, 1927
Iliac crests	..	Buchman, 1925
Upper end of tibia	..	Ritter, 1929
Astragalus	..	Monchet, 1928
Accessory patellar centers	..	Larsen, 1921
First metatarsal	..	Wagner, 1930
Head of second metatarsal	..	Freiberg, 1914
Head of fifth metatarsal	..	Iselin, 1912
Vertebral epiphyses	..	Scheurman, 1921
Vertebral body	..	Calvé, 1926
Sternal end of clavicle	..	Friedrich, 1924
Internal condyle of humerus	..	Legg, 1930; Löhr, 1930
Carpal scaphoid	..	Preiser
Medial sesamoid of first metatarsal	..	Renander, 1924
Os tibiale externum	..	Haglund, 1908
Ischiopubic junction	..	Odelberg, 1924; Van Neck, 1924

1. Engelbach, William: Endocrine Medicine, Springfield, Ill., C. C. Thomas, 1932.

2. Engelbach, William, and Schaefer, R. L.: Endocrine Dwarfism, J. A. M. A. **103:** 464-468 (Aug. 18) 1934. Engelbach, William; Schaefer, R. L., and Brosius, W. L.: Endocrinology **17:** 250-262 (May-June) 1933. Engelbach, William, and Schaefer, R. L., ibid. **18:** 387-392 (May-June) 1934. Schaefer, R. L., ibid. **20:** 64-71 (Jan.) 1936.

in this developing osseous structure. There were only three patients in the entire group who had any symptoms referable to the osseous involvement; two were referred by the orthopedic collaborator and one by a pediatrician,[3] who recognized the possibility of an associated endocrine factor as the cause of the chondro-epiphysitis. It is well to emphasize here that all the

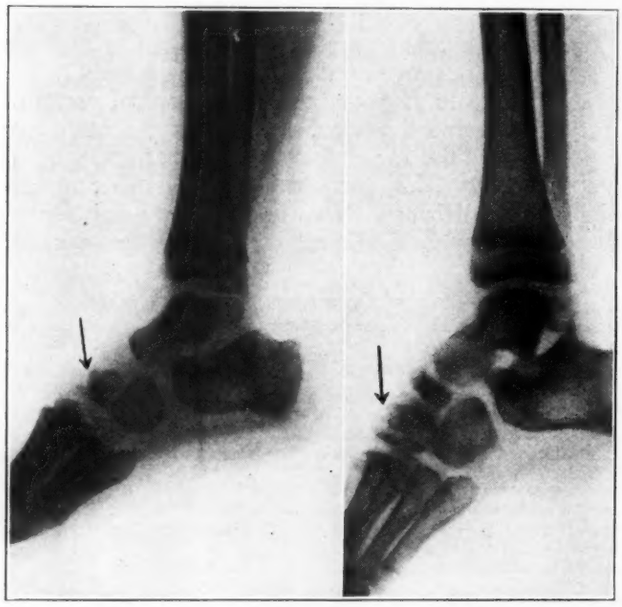

Fig. 1.—Left: chondro-epiphysitis of tarsal scaphoid (Köhler's disease); right: after fifteen months' treatment with thyroid extract at tolerance.

patients in this group receive a careful routine diagnostic survey including history, physical examination and laboratory studies such as confirming basal metabolic

TABLE 2.—*Incidence of Chondro-Epiphysial Disturbance in Endocrinopaths: Age Group 8 to 15 Years*

		Cases Showing Chondro-Epiphysial Disturbance		Extent of Epiphysial Involvement		
	Number Studied	Number	Per Cent	One Epiphysis	More Than One Epiphysis	Per Cent Showing Multi-epiphysial Involvement
Endocrinopaths.....	258	91	35.2	53	38	41.7
Patients with evidence of hypothyroidism....	218	85	39.0	49	36	42 3
Patients without ascertained evidence of hypothyroidism.....	40	6	15.0	4	2	33.3

rates, urinalysis, blood count and blood chemistry studies including sugar, nonprotein nitrogen, cholesterol, calcium and phosphorus.

Our control group study was made possible at the Anatomical Laboratory and Associated Foundations of Western Reserve University [4] through the courtesy of the late Dr. T. Wingate Todd.

The records of ninety-nine children who fulfilled our requirements as being free from endocrine imbalance were placed at our disposal for study. The age range was the same as of the endocrinopaths studied. These children are referred to the foundation for study only after they have been certified by their family physician or pediatrician as being free from gross physical or mental defect and from behavioristic problems. No particular type of clinical examination is designated, but the physician is required to ascertain that the child is in a good state of organic health and shows no clinical evidence of metabolic defect. Roentgenographic studies with a view of bone age assessment are made as a routine at yearly intervals, or oftener if the child is under 5 years. X-ray films of six areas are made: the hand, elbow, shoulder, hip, knee and ankle. The majority of the files consulted showed at least four yearly studies entailing twenty-four x-ray films; a few had had seven studies a year or a total of forty-two films, and it was decidedly in the minority that there were only one or two studies a year. All areas are roentgenographed in the anteroposterior and lateral positions. An approximate total of 2,200 films was viewed.

Of the group reviewed only seven, or 7 per cent, showed evidence of chondro-epiphysitis.

At this time our studies are not sufficiently advanced to give any imposing factual data with regard to follow-up x-ray films after treatment. However, in eight cases so far studied a complete return to normal bone detail roentgenographically has been obtained by the administration of a fully tolerant and maintained dose of thyroid extract. Zuck [5] in 1933 reported that the restorative process could be hastened by endocrine therapy. That normality can result without treatment is readily admitted, as we are aware that not infrequently mild endocrine imbalances spontaneously correct themselves. It is suggested, however, that such x-ray evidence is a definite indication for thyroid therapy.

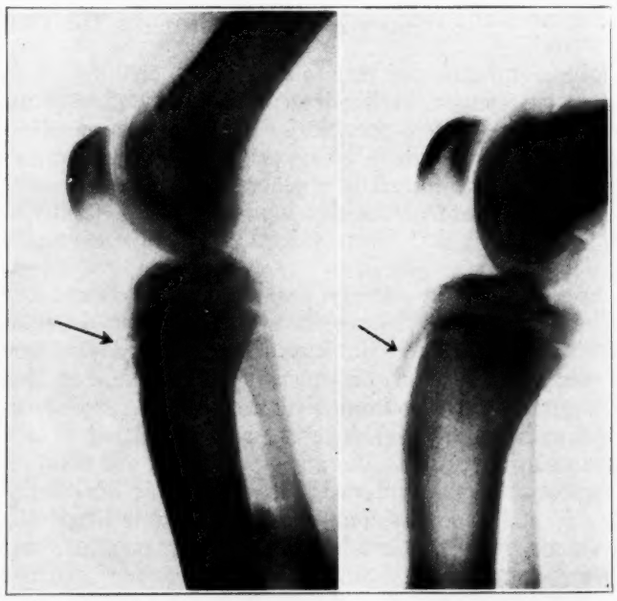

Fig. 2.—Left: chondro-epiphysitis of the tibial tubercle; right: after twelve months' treatment with thyroid extract at tolerance. Note improvement in the bony detail of the epiphysis and disappearance of the fragmentation and irregularity in both cases.

CONCLUSION

1. A group of 258 endocrinopathic patients in an age range of 8 to 15 years was studied: ninety-one, or 35.2 per cent, displayed single or multiple evidence of chondro-epiphysitis; eighty-five of the ninety-one showed clinical or laboratory evidence of primary or secondary hypothyroidism.

3. Dr. C. L. Douglas of Detroit.
4. Aid was received from Mr. Howard H. Gradis, Dr. Todd's assistant.
5. Zuck, T. T.: J. Pediat. **3**: 424-433 (Sept.) 1933.

2. A control group of ninety-nine nonendocrine patients in the same age range showed only seven, or 7 per cent, cases in which there was involvement of chondro-epiphysial structures.

3. Chondro-epiphysitis is of endocrine origin, specifically due to a primary or secondary hypothyroidism, usually asymptomatic and, to a degree, undoubtedly generalized.

4. Chondro-epiphysitis is a pathognomonic sign of hypothyroidism.

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DIPHTHERIA IMMUNITY IN CHICAGO

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Although diphtheria mortality and morbidity have been gradually decreasing in most parts of the United States for the past twenty-five years, they have not been reduced to the level which it was hoped would be attained. Antitoxin, control of carriers and the Schick test were important. The discovery of toxoid added new impetus to the efforts to control this disease. It was believed that the inoculation of a large proportion of the child population would result in almost complete eradication of diphtheria.

The results obtained with the use of toxoid did not, however, approximate expectations. The present study explains to some extent this failure.

In Chicago, until 1931, three 1 cc. doses of antitoxin at weekly intervals were used for diphtheria immunization. In 1931 plain toxoid, two 1 cc. doses at weekly intervals, was employed. From 1932 to 1934 alum toxoid, two 1 cc. doses at weekly intervals, was used; from June 1934 to August 1934 a 1 cc. dose of alum precipitated toxoid was used; from Aug. 1, 1934, to January 1935, two 1 cc. doses of alum toxoid at weekly intervals were given; from January 1935 until late in 1936 a 1 cc. dose of alum precipitated toxoid was again used. Since that time we have employed three doses of plain toxoid given a month apart, the first dose 0.5 cc. and the other two doses 1 cc. each.

Before a change was made from one type of preparation to the other, experimental tests were carried out on groups of at least 1,000 children each. The children were inoculated with the various preparations, and Schick tests were made at intervals of from two to six months to determine whether protection against diphtheria had been obtained. In other words, the determination of the type of preparation to be used was based on the Schick testing of inoculated children, together with evidence obtained from similar studies carried out elsewhere.

Table 1 reveals that diphtheria mortality and morbidity reached a low level in 1933. After that time there was a gradual rise. The levels, however, did not nearly approach those of the years prior to 1933. The reduction in 1933 was the result of widespread inoculation with toxoid of children in the city of Chicago.[1] Since that time the number of inoculations each year have been at least 80 per cent of the number of children born.

We therefore believe that, as far as the number of children inoculated is concerned, we are taking the steps necessary to control diphtheria adequately. Failure to maintain the low rates of mortality and morbidity obtained in 1933 must consequently be ascribed to the fact either that inoculated children are not rendered immune or that if they do become immune this immunity is lost with the passage of varying intervals of time.

In order to determine what has been occurring with the groups of children inoculated with the various preparations employed in Chicago, a study was undertaken in 1936. Children inoculated with these different preparations were selected of as nearly the same age as possible. The ages at the time of testing varied generally between three and seven years. In the group that received toxin-antitoxin, the children were somewhat older, that is between 9 and 13 years. Those who received three doses of toxoid at monthly intervals for the most part were between 1 and 2 years of age. A record was kept of the intervals between the time the inoculation for the prevention of diphtheria was given and the time at which blood was drawn for the determination of the antitoxin content. Antitoxin titrations were determined by the Fraser modification of Romer's technic,[2] except that white guinea pigs were used instead of rabbits. The children were given a Schick test immediately after blood was drawn for the antitoxin determination. This was done because the Schick test in itself is known to stimulate the formation of antitoxin in the blood.

TABLE 1.—*Diphtheria Mortality Rate per Hundred Thousand of Population*

Years	Cases	Deaths	Death Rate
1912	7,288	953	41.4
1913	8,593	968	41.1
1914	7,071	767	31.8
1915	5,863	679	27.6
1916	6,980	787	31.3
1917	10,290	1,229	47.8
1918	5,708	721	27.5
1919	6,361	592	22.1
1920	7,761	630	23.1
1921	9,313	678	24.4
1922	7,367	564	19.9
1923	5,836	366	12.7
1924	3,672	216	7.4
1925	2,926	241	8.1
1926	2,540	224	7.2
1927	4,123	442	13.8
1928	4,880	457	14.0
1929	6,012	513	15.4
1930	4,962	411	12.1
1931	3,146	215	6.2
1932	1,266	68	1.9
1933	154	9	0.3
1934	327	41	1.2
1935	762	83	2.4
1936	614	87	2.5
1937	655	84	2.3

RESULTS

Group 1. The children in this group had received three injections of toxin-antitoxin a week apart during 1930. The children ranged in age from 3 to 7 years at the time of the injection, the majority being 5 years of age. The ages at the time the Schick tests were done ranged from 9 to 13 years. In the majority of

1. Bundesen, H. N.; Fishbein, W. I., and Niblack, H. C.: Diphtheria Control in Chicago, J. A. M. A. **100**: 1093 (April 8) 1933.

2. Fraser, D. T.: Tr. Roy. Soc. Canada (Sect. V, Biol. Sc.) **25**: 175 (May) 1931.

instances the blood was drawn and the Schick test done about six years after inoculations had been given.

The Schick tests gave negative results in all except two cases. Both of these children were 5 years of age at the time of the inoculation and 11 years of age at the time the Schick test was done. Thus, of 125 total children in the group, 123 showed negative Schick tests.

Table 2 shows the results of the antitoxin determination of these 123 Schick-negative children. Of the two children who showed positive Schick tests, one had less than $\frac{1}{500}$ unit of antitoxin per cubic centimeter of blood, while the other had between $\frac{1}{500}$ and $\frac{1}{250}$ unit of antitoxin.

Group 2. This group was made up of children who had been given plain toxoid—two doses at weekly intervals—during 1932. The children were about 5 years of age at the time of inoculation and about 9 years of age at the time the blood was drawn and the Schick test done.

There were 125 children in this group; 106 were Schick negative and nineteen Schick positive. Of these nineteen Schick-positive children twelve had less than $\frac{1}{500}$ unit of antitoxin per cubic centimeter of blood, four had between $\frac{1}{500}$ and $\frac{1}{250}$, two had between $\frac{1}{250}$ and $\frac{1}{100}$ and one had between $\frac{1}{100}$ and $\frac{1}{50}$.

Group 3. This group consisted of children who were given two doses of alum toxoid, not precipitated, one week apart. These children were inoculated between 1932 and 1934. Their average age at the time of inoculation was between 5 and 6 years. The blood was drawn and the Schick test done when the children were between 7 and 9 years of age.

Of these 145 children, sixteen showed positive Schick tests and 129 showed negative Schick tests. Of those who showed positive Schick tests, seven had less than $\frac{1}{500}$ unit of antitoxin per cubic centimeter of blood and nine had between $\frac{1}{500}$ and $\frac{1}{250}$ unit. In table 2 under group 3 are the results of the antitoxin determination in the Schick-negative children.

Group 4. In this group there were 119 children, ranging in age from 5 to 7 years. This group had been given one dose of alum precipitated toxoid in 1934. Blood was drawn and Schick tests were done in 1936.

Of these 119 children, six showed positive Schick tests and 113 were negative. Of those who showed positive Schick tests, three had between $\frac{1}{500}$ and $\frac{1}{250}$ unit of antitoxin per cubic centimeter of blood, two had less than $\frac{1}{500}$ and one had between $\frac{1}{25}$ and $\frac{1}{10}$. The antitoxin determinations and percentages of those who showed negative Schick reactions are seen under group 4 in table 2.

Group 5. In the latter part of 1936 and in 1937 a group of 107 children were given plain toxoid, three doses at monthly intervals. The ages of the children varied from 8 months to 2 years at the time of inoculation. Blood was drawn for antitoxin determination and the Schick test was done on this group in the early part of 1938, which was from one to one and one-half years after the inoculations were given.

In this entire group of 107 young children there were none who showed positive Schick test reactions—all were Schick negative. The antitoxin determination of these children can be seen under group 5 in table 2. Schick test studies were carried out on a large number of the children in each of the five groups during 1936, 1937 and 1938.

In group 1, in 1936 and 1937, Schick tests were done on a group of 583 children ranging in age from 10 to 15 years, who were given antitoxin, three doses a week apart, prior to 1931. Of this group of 583 children 546, or 93.6 per cent, showed negative Schick test reactions and thirty-seven, or 6.4 per cent, were positive.

In group 2, Schick tests were done on a total of 829 children ranging in age from 10 to 15 years, who had received two doses of plain toxoid one week apart in 1931 and 1932. Of these children 702, or 87.1 per cent, showed negative Schick test reactions and 127, or 12.9 per cent, positive.

In group 3, Schick tests were done in 1936 and 1937 on 634 children ranging in age from 7 to 9 years. These children received two doses of 0.2 per cent alum toxoid, not precipitated, one week apart in 1932 to 1934. Of these 514, or 81.1 per cent, showed negative Schick test reactions and 120, or 18.9 per cent, positive.

In group 4, a total of 660 children ranging in age from 1 to 4 years, were given one dose of alum precipitated toxoid in 1934. Of this group, in which Schick tests were done in 1936 and 1937, 612, or 92.7 per cent, showed negative results, and forty-eight, or 7.3 per cent, positive results.

In group 5, 1,000 children, or 100 per cent, of those tested, during 1938, ranging in age from 1 to 11 years, but for the most part from 1 to 2 years of age, responded negatively to the Schick test. This group was given plain toxoid, three doses a month apart, during 1936 and 1937.

TABLE 2.—*Diphtheria Antitoxin Content of Children with Negative Schick Reactions*

Units of Diphtheria Antitoxin per Cc. of Blood Serum	Group 1		Group 2		Group 3		Group 4		Group 5	
	Number of Children	Per Cent of Total Specimens	Number of Children	Per Cent of Total Specimens	Number of Children	Per Cent of Total Specimens	Number of Children	Per Cent of Total Specimens	Number of Children	Per Cent of Total Specimens
1: 10 or more	78	63.4	53	50.0	62	48.1	33	29.2	79	73.8
1: 25 to 1: 10	14	11.4	14	13.2	19	14.7	16	14.2	17	15.9
1: 50 to 1: 25	8	6.5	9	8.5	7	5.4	16	14.2	8	7.5
1:100 to 1: 50	7	5.7	9	8.5	14	10.9	15	13.3	1	0.9
1:250 to 1:100	4	3.3	7	6.6	10	7.8	17	15.0	2	1.9
1:500 to 1:250	5	4.1	6	5.7	7	5.4	12	10.6	0	0
Less than 1:500	7	5.7	8	7.5	10	7.8	4	3.5	0	0
Total	123		106		129		113		107	

Group 1: Toxin-antitoxin 1 week, 3 doses.
Group 2: Plain toxoid, 1 week, 2 doses.
Group 3: Toxoid 0.2 per cent alum added, 1 week, 2 doses (not precipitated).
Group 4: Alum precipitated toxoid, 1 dose.
Group 5: Plain toxoid, 3 doses, monthly intervals.

COMMENT

Immunity to diphtheria may occur in various ways. It may be inherited, it may be acquired as a result of inoculation of some type of antigenic material or it may develop as a result of the inhalation of minimal

doses of diphtheria organisms. There is the possibility of "maturation immunity" or insusceptibility to disease due to maturity, independent of minimal inoculations.

In most children who are immune to the disease such immunity has probably developed as a result of all these factors. The inherited immunity is probably almost completely lost, in practically all cases, by the time the child has reached 1 year of age. Immunity that develops as the result of the inoculation of antigenic material lasts for a varying period of time, depending on the type of material inoculated and on the interval between doses. The longer the period after inoculation, the less should be the antitoxin content of the blood if these children are not exposed from time to time to diphtheria organisms, so that further immunity may develop as a result of the natural body processes.

In a consideration of the results which we have obtained in our experimental work, these various factors must be borne in mind. A greater interval elapsed between the time of inoculation and the time of testing in the children in group 1 than in the other groups. Hence from this standpoint it is to be expected that they would show lesser amounts of antitoxin. However, there was a greater opportunity for natural immunity to develop. Therefore an exact comparison is not possible between the children in the various groups. Nevertheless the figures seem to indicate that, both from the standpoint of the amount of antitoxin in the blood and from the standpoint of the Schick test, those children who received three doses of toxoid a month apart were better protected against diphtheria than those who received other types of inoculations. Just how long this immunity will continue cannot be stated at the present time. It is our intention to carry out further tests of the antitoxin content of the blood, from year to year, on the same group to determine the answer to this question.

Evaluation of the methods used to immunize the children in the five groups cannot be given with exactness till more time has passed and the relapse rate in group 5 can be determined. The observation may be made, however, that the time interval between immunization and the Schick test decreased by the integral variant of 1, while the percentage of relapses decreased by roughly 6 per cent a year from group 2 to group 5, giving an expected relapse rate in group 5 of zero, which it actually was. This does not make the results of a relapse rate of zero seem as good an omen of future high Schick negatives as may be expected from first examining the results. In fact, group 1, with a time lapse between immunization and Schick testing of six years and a relapse rate of only 2.4 per cent, has the statistical advantage over all other groups provided the important effect of environmental stimuli can be discounted.

Jensen [3] has experimentally shown that the rate at which antitoxin disappears from the blood is a function of a rectangular hyperbola: $Y = \frac{1}{X}$ or

$$T = \frac{A - X}{AX} \cdot \frac{1}{K}$$

A is maximum concentration of antitoxin following an antigenic stimulus, X is the amount of antitoxin which remains, T units of time after the moment at which A is reached, K is a constant which is different for each individual. This is the same formula by which lysins and other antibodies disappear from the blood.

3. Jensen, Claus: Deutsche med. Wchnschr. 57: 324 (Feb. 20) 1931.

One can see by the formula that when the amount of antitoxin is small the rate of decrease is small; i. e., if a patient has 1 unit of antitoxin per cubic centimeter of blood, his rate of loss of antitoxin is 10,000 times greater than when the antitoxin reaches 0.01 unit per cubic centimeter. If X or the antitoxin level is small and K is small, very long periods of time may elapse before an appreciable loss of antitoxin has been effected.

The toxin-antitoxin courses took place, however, at a time when the exposure to endemic diphtheria was much greater than could be experienced by the other groups; hence, so many of this first group encountered adequate secondary stimuli that the frequency of the Schick positive reactions decreased.

The main value of repeated doses of an antigen is that, should the antitoxin titer fail to rise within two or three days after the first inoculation it will not rise until the advent of a secondary stimulus.

The reasons given for failure to maintain the low rates of mortality and morbidity—that inoculated children are not rendered immune or that this immunity is lost with the passage of varying intervals of time—are ruled out by figures given in tables which show that

TABLE 3.—*Diphtheria Antitoxin Content of Blood in Inoculated Children.*

Units of Diphtheria Antitoxin per Cc. of Blood Serum	Positive Reaction to Schick Test by Groups Number of Children Reacting in				
	Group 1	Group 2	Group 3	Group 4	Group 5
1: 10 or more	..	..	..	..	..
1: 25 to 1: 10	..	..	..	1	..
1: 50 to 1: 25	..	..	..	..	..
1:100 to 1: 50	..	1	..	..	..
1:250 to 1:100	..	2	..	..	..
1:500 to 1:250	1	4	9	3	..
Less than 1:500	1	12	7	2	..
Total	2	19	16	6	0

the inoculated children are immune and that the immunity does last for a long period of time. Some other reason for this increase must be looked for.

The idea, backed by experimental proof, has been repeatedly put forward that the number of carriers of virulent diphtheria organisms is increased in proportion to the number of Schick negative persons in a community, and when a Schick positive is replaced by a Schick negative a potential avirulent carrier is replaced by a potential virulent carrier. Thus with a declining avirulent carrier rate goes a corresponding increase in the virulent carrier rate.

"Incomplete artificial immunization of a group may have no inhibitory effect on the total incidence of diphtheria and, for a short period, may increase the risk of attack in the unprotected section of the community." [4] The increased number of virulent carriers (gravis), coupled with the fact that an increased rate of relapses occurred in groups 2, 3 and 4, supposedly due to failure of the action of natural environmental stimuli, account in part at least for the upswing of diphtheria morbidity and mortality rates.

In the past, the Schick test has been relied on almost exclusively as an index of the immunization status against diphtheria. The Schick test is subject to some criticism. Presumably the test is negative when there is more than $\frac{1}{250}$ unit of antitoxin per cubic centimeter of blood.

4. Dudley, S. F.; May, P. M., and O'Flynn, J. A.: Special Report Series, No. 195, Medical Research Council, London, 1934.

It is interesting to note that fifty-nine of the 471 children in the first four groups studied had a negative Schick test with an antitoxin content of blood less than $\frac{1}{250}$ of a unit per cubic centimeter.

It has been stated that a child should have at least $\frac{1}{25}$ unit of antitoxin per cubic centimeter of blood to be relatively well protected against diphtheria. Hence the unreliability of the Schick test as the measure against diphtheria is indicated.

We find that, in group 1, 74 per cent of the children who received toxin-antitoxin had more than $\frac{1}{25}$ unit. In group 2, 63.2 per cent of the children who had received two doses of plain toxoid had more than this quantity. In group 3, 62.8 per cent of those who had received two doses of plain alum toxoid a week apart had more than this amount. In group 4, 43.4 per cent of the children who had received one dose of alum precipitated toxoid had more than this amount, while, in group 5, 89.7 per cent of the children who had received three doses of plain toxoid had more than $\frac{1}{25}$ unit of antitoxin per cubic centimeter of blood serum.

TABLE 4.—*Procedures Employed by One Hundred and Four State and City Health Departments*

Doses Employed	Number of City and State Health Departments
Alum Precipitated Toxoid (The dose varies from 0.5 to 1 cc.)	
1	58
2 (1 month apart)	9
2 (2 weeks apart)	3
2 (3 weeks apart)	2
2 (1 week apart)	1
2 (2 months apart)	1
2 (1 year apart)	1
Plain Toxoid (The dose varies from 0.5 to 1.5 cc., the usual dose being 1 cc.)	
2 (1 month apart)	10
2 (3 weeks apart)	9
2 (2 weeks apart)	5
3 (1 week apart)	5
3 (3 weeks apart)	5
3 (2 weeks apart)	3
3 (1 month apart)	3
Toxin-Antitoxin (Employed in three doses a week apart in five city or state health departments)	

Of the methods of inoculation that have been employed in these five groups of children, it seems, from our figures, that without question the use of three doses of plain toxoid a month apart is the most efficient.

Further studies, however, will be necessary in order to determine the duration of such immunity as is acquired.

At present, according to the replies to a questionnaire which we sent to the health officers of many states and cities, there is great variation in the procedures employed in an attempt to produce immunity against diphtheria. Replies were received from 104 state and city health departments: thirty-six state and sixty-eight city. These replies indicated that thirty-seven cities employed alum precipitated toxoid almost exclusively, twenty-two employed plain toxoid almost exclusively, and seven employed both preparations, while two used both alum precipitated toxoid and toxin-antitoxin. The alum precipitated toxoid, according to these replies, is used more often than any type of antigen, the one dose method being employed by fifty-eight city and state health departments. The dose varied from 0.5 to 1 cc.

Table 4 indicates the number of health departments using alum precipitated toxoid, plain toxoid and toxin-antitoxin.

CONCLUSION

The evidence seems to indicate that more immunity to diphtheria is obtained when the antigen is given in a greater number of doses and the interval between inoculations is longer. Three doses of plain toxoid at monthly intervals seem to give the best result.

The Schick test cannot be relied on as a definite measure of the immunization status. The type of inoculations being used throughout the United States at present for the most part cannot be relied on to produce permanent immunity. With the adoption of three inoculations of toxoid at monthly intervals, it is to be hoped that diphtheria mortality and morbidity rates will show an even greater reduction in the future than they have in the past.

ADDENDUM

In March and April 1939, since the completion of this paper, seventy children in group 5 were retested to determine the antitoxic content of the blood and their reactions to the Schick test.

All the children were Schick negative. Thirty-eight, or 55.8 per cent, had more than $\frac{1}{10}$ unit of antitoxin per cubic centimeter of blood; seventeen, or 25.0 per cent, from $\frac{1}{25}$ to $\frac{1}{10}$; nine, or 13.2 per cent, from $\frac{1}{50}$ to $\frac{1}{25}$; three, or 4.4 per cent, from $\frac{1}{100}$ to $\frac{1}{50}$, and one, or 1.4 per cent, from $\frac{1}{250}$ to $\frac{1}{100}$. These figures represent a loss of 18.0 per cent in the group having more than $\frac{1}{10}$ unit, a gain of 9.1 per cent in those having from $\frac{1}{25}$ to$\frac{1}{10}$, a gain of 5.7 per cent in those having from $\frac{1}{50}$ to $\frac{1}{25}$, and of 3.5 per cent in those having from $\frac{1}{100}$ to $\frac{1}{50}$, with a loss of 0.4 per cent in those having from $\frac{1}{250}$ to $\frac{1}{100}$ unit.

These tests were carried out approximately two to two and one-half years following the administration of three doses of toxoid at intervals of one month.

Of the children who received three doses of plain toxoid at intervals of a month, 80.8 per cent have maintained, for a period of from two to two and one-half years, an antitoxic content above $\frac{1}{25}$ unit, which is the amount stated to be necessary for relatively good immunity against diphtheria. Thus in group 5 about 9 per cent of the children in a period of a year shifted from more than $\frac{1}{25}$ unit to less than this amount. At present none of this group have less than $\frac{1}{250}$ unit of antitoxin, which is the amount stated to be necessary to give a negative Schick test. These figures would indicate that with three doses of plain toxoid, given a month apart, immunity is relatively well maintained for a period of at least two to two and one-half years.

The children in this group probably have not had as great a possibility of coming in contact with diphtheria as those in the other groups. It seems evident, however, that this group of children must be retested yearly for the next several years before it will be possible to state definitely the rate at which immunity, as determined by the antitoxic content of the blood, is lost from year to year.

It is of interest to note that ten children of group 5 were retested in 1939 for their antitoxic content in addition to the seventy referred to. These ten, however, were Schick tested about three weeks prior to the drawing of blood for the antitoxin determination. Eight of these ten had more than $\frac{1}{10}$ unit in 1937; one had $\frac{1}{50}$ and the other $\frac{1}{25}$ unit. In 1939, after the Schick

test, eight showed more antitoxin than they had in 1937, one had the same amount, and one showed a slightly lesser amount.

Thus it seems from these figures that the stimulation of a Schick test in itself probably may be sufficient to increase the antitoxin content of the blood at least temporarily.

In view of the fact that there seems to be a gradual loss in the antitoxic content of the blood, consideration should be given to the advisability of reinoculating children with one dose of some antigen at intervals of from three to five years to maintain a sufficiently high level of antitoxin to ward off the disease.

GALLBLADDER DISEASE IN PATIENTS UNDER THIRTY YEARS OF AGE

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While disease of the gallbladder occurs at any age, even in fetuses and newly born infants,[1] it is considered primarily a disease of middle age and later life.[2] Approximately 16.5 [3] to 21 [4] per cent of patients operated on for symptoms due to disease of the gallbladder are under 30. In an effort to determine the course of the disease before 30 and the end results following cholecystectomy, an analysis was made of sixty-three cases of operation performed during the first three decades of life. These cases represented 21 per cent of 300 consecutive cases in which I had performed the operation.

There were fifty-eight females and five males. The distribution by decades and by sex is shown in table 1.

The ratio of females to males was 12 to 1. This figure is much higher than the ratio for all ages, for which reported values have varied between 2.05 to 1 [3] and 5.5 to 1.[5] In the group of 300 cases from which these sixty-three were selected, the ratio was 5.2 to 1. On the other hand, in a study limited to 432 children under 15 years of age [6] the ratio, while still in favor of females, was less than 2 to 1.

The general belief that a patient with disease of the gallbladder is usually overweight is not always borne out by the facts. Doran and his associates,[7] for example, reported that 53 per cent of their patients were overweight; Smithies,[3] on the other hand, reported but 8 per cent obese or consistently gaining weight. In the series of patients under consideration, twelve (19 per cent) were definitely overweight, forty-seven (74.6 per cent) were of average weight and four (6.3 per cent) were noticeably thin.

Since symptoms referable to the gallbladder occur frequently in relation to childbirth, it was not surprising that of the forty-eight married women in the series twenty-three (47.7 per cent) had symptoms either during pregnancy or shortly thereafter. In two cases the symptoms were so severe and persistent that operation was done during pregnancy; the pregnancy continued uneventfully. The remaining twenty-one patients deferred operation for at least two years, but all were operated on within five years of the onset of symptoms.

Acute infectious diseases, particularly typhoid fever and scarlet fever, have been frequently cited as etiologic factors in disease of the gallbladder. In this group thirty-five (55.5 per cent) gave a history of one or more infectious diseases. Twenty-eight patients had had measles, eight scarlet fever and four typhoid fever. Of the twenty-three patients with symptoms during pregnancy, only nine (39.1 per cent) gave a history of acute infection. This would imply that the likelihood of disease of the gallbladder in pregnant women is not increased by a previous infectious disease.

The symptoms were similar to those presented by older patients, such as pain in the area of the gallbladder at times radiating to the back and sometimes to the shoulders, and belching, nausea and vomiting. While pain was always the prominent symptom, in fifty cases (79.3 per cent) it was severe enough to be described as colic. In fourteen cases (22.2 per cent) it frequently occurred after eating. Only fifteen patients (23.8 per cent) complained of eructations, although belching was reported [3] as "prominent and distressing" in as many as 68.9 per cent of a group of patients of all ages. Nausea and vomiting affected twenty-nine (46 per cent), occurring almost as frequently as in older patients.

The symptoms varied in duration from twenty-four hours, when the patient was operated on after the first attack of colic, to twelve years in a patient of 29.

TABLE 1.—*Sex and Age by Decades*

Age (years)	Males	Females	Total
To 9	1	0	1
10 to 19	0	4	4
20 to 29	4	54	58
Totals	5	58	63

TABLE 2.—*Duration of Symptoms*

Years	Patients
Less than 1	19
1 to 2	18
2 to 3	16
4	5
5	3
More than 5	2

Table 2 shows the duration of symptoms for these sixty-three patients. The average duration for the entire group was 2.1 years and the average age was 24.7 years.

While only five patients (7.9 per cent) were operated on before the age of 20 (table 1) four additional patients, making a total of nine (14.2 per cent), had symptoms begining in their teens. Potter [6] collected from the literature 432 cases under the age of 15 years, and Starr [8] noted that 36 per cent of his patients had symptoms before the age of 25.

But five patients (7.9 per cent) showed evidence of clinical jaundice, and three of these (60 per cent) had no stones in the gallbladder. Blalock [9] pointed out that

1. Shawan, H. K., and Long, E. C.: Gallbladder Disease in Young Children, Am. J. Surg. **21:** 43 (July) 1933. Hamilton, H. B.; Rich, C. O., and Bisgard, J. D.: Cholecystitis and Cholelithiasis of Childhood, J. A. M. A. **103:** 829 (Sept. 15) 1934.
2. Niemeier, O. W.: The Importance of Earlier Operation in Chronic Gallbladder Disease, Canad. M. A. J. **37:** 332 (Oct.) 1937. Smithies.[3] Blalock.[9]
3. Smithies, Frank: Clinical Manifestations in Gallbladder Disease: A Study of 1,000 Operatively Demonstrated Cases, Northwest Med. **19:** 31 (Feb.) 1920.
4. Deaver, J. B., and Bortz, E. L.: Gallbladder Disease, J. A. M. A. **88:** 619 (Feb. 26) 1927.
5. Wilson, W. D.; Lehman, E. P., and Goodwin, W. H.: Prognosis in Gallbladder Surgery, J. A. M. A. **106:** 2209 (June 27) 1936.
6. Potter, A. H.: Biliary Disease in Young Subjects, Surg., Gynec. & Obst. **66:** 605 (March) 1938.
7. Doran, W. T.; Lewis, K. M.; Deneen, E. V., and Hanssen, E. C.: Gallbladder Surgery: A Report of 200 Consecutive Operated Cases of Gallbladder Disease, Ann. Surg. **98:** 321 (Sept.) 1933.
8. Starr, F. N. G., cited by Potter.[12]
9. Blalock, Alfred: A Clinical Study of Biliary Tract Disease, J. A. M. A. **83:** 2057 (Dec. 27) 1924.

jaundice was almost as common in patients of all ages with noncalculous cholecystitis as in patients with cholelithiasis.

Of the sixty-three gallbladders that were removed, four (6.3 per cent) were acutely inflamed and fifty-nine (93.6 per cent) showed chronic cholecystitis, including one with cholesterosis. Thirty-eight gallbladders with chronic inflammation (64 per cent) contained stones, and stones were also present in all but one of the acutely inflamed gallbladders. In other words, a total of forty-one gallbladders (65 per cent) had stones. One patient, a boy of 7 years, showed noncalculous acute cholecystitis.

Besides this 7 year old boy there were twenty-one patients with noncalculous cholecystitis; sixteen of these (76.1 per cent) had colicky pain. Other investigators [10] have reported that approximately 55 per cent of their patients with noncalculous chronic cholecystitis gave a history of colic.

Eight common ducts (12.6 per cent) were opened and, together with the hepatic ducts, explored. Only three common ducts (37.5 per cent) contained stones. The patients with choledocholithiasis had no jaundice at any time. They were 24, 27 and 29 years old. The shortest duration of the disease was four years, in the 24 year old patient. Symptoms were present for five years in the 29 year old patient and for seven years in the 27 year old patient.

The incidence of stones in the common duct in the entire series was 4.7 per cent, but for the forty-one cases of calculous gallbladder the incidence was 7.3 per cent. It has been reported that in only 4 per cent of cases of noncalculous gallbladder are stones found in the common duct.[11] In the twenty-one instances of noncalculous gallbladder in this series no stones were present in the biliary ducts.

TABLE 3.—*Comparison of End Results*

Author	Cured or Improved: With Stones, %	Cured or Improved: Without Stones, %
Present study (patients under 30)	97.2	85.0
Wilson, Lehman and Goodwin [5]	79.0	64.0
Kunath (J. A. M. A. **109:** 183 [July 17] 1937)	84.3	69.0
Graham, E. A., and Mackey, W. A. (J. A. M. A. **103:** 1497 [Nov. 17] 1934)	....	65.2
Brown, M. J. (Am. J. Surg. **41:** 238 [Aug.] 1938)	....	70.8

Stones are not rare in the common and in the hepatic duct of patients under 30. Of 221 patients with stones in the biliary ducts, fifteen (7.4 per cent) were under 30.[11] Moreover, of 226 patients under 15 with disease of the gallbladder, thirteen (5.7 per cent) had stones in the biliary ducts, including an 8 month fetus and two infants of 12 and of 25 days.[12] On the other hand, the incidence of stones in the common and in the hepatic duct of patients of all ages has been reported as high as 21.2 per cent.[11]

One patient in this group had acute hemorrhagic pancreatitis associated with cholelithiasis. She was an obese woman 29 years old who had had symptoms for three years before operation.

There were no deaths in this series.

Only four patients had any postoperative complications. One patient had phlebitis, which kept her in the hospital thirty-four days; she had had a similar complication following childbirth three years previous to operation. One patient had a superficial infection of the operative wound, which cleared up in several days. Another patient, aged 29 years, whose common duct was opened and explored and the sphincter of Oddi dilated, had a gas bacillus infection. The infection was not severe and she was discharged from the hospital as "well" on the twenty-first postoperative day.

It was possible to follow fifty-seven patients (90.4 per cent) after operation. Twenty-four patients were followed from one to four years, fifteen were seen from five to eight years after cholecystectomy and eighteen were followed from eight to ten years or more. The longest period of observation was thirteen years.

Fifty-three patients (92.9 per cent) were free from symptoms when last seen. Thirty-six (97.2 per cent) of the thirty-seven who had gallstones were entirely relieved; seventeen (85 per cent) of the twenty who had noncalculous chronic cholecystitis were either greatly improved or completely relieved. These satisfactory end results were much better than those reported for patients of unclassified age groups (table 3). In the reports for cholelithiasis the improvement varied from almost 3 to 18 per cent, while for noncalculous cholecystitis the improvement ranged from 14 to 21 per cent.

Four patients (7 per cent) had attacks of gallbladder colic following cholecystectomy. One of these patients had cholelithiasis and three had noncalculous cholecystitis. The pain was severe enough in two cases to warrant repeated hypodermic injections of morphine by the family physician. At no time did any of these patients have jaundice, chills or fever. X-ray examination showed no abnormality. Whether these postoperative attacks of colic were due to overlooked stones in the common duct, to biliary dyskinesia or to some extrabiliary abnormality is problematic.

One patient who was considered cured was free from symptoms for two years after removal of a noncalculous gallbladder. Then the attacks recurred and persisted for three years. Because nonsurgical treatment gave no relief, another operation was done; only adhesions were found. Since then she has been free from all symptoms.

In any group of patients with a noncalculous gallbladder the question may be raised whether operation is justified, in view of the large percentage that are unrelieved. Blackford and his associates [13] followed 200 patients of all ages with noncalculous chronic cholecystitis for from five to fifteen years. They reported that but 37 per cent had satisfactory relief without operation for more than eight years; 48 per cent who had nonsurgical treatment for the same period either were operated on later or should have been, because of persistence of symptoms.

SUMMARY

Sixty-three patients operated on for disease of the gallbladder before the age of 30 represented 21 per cent of 300 consecutive cholecystectomies.

Operation before the age of 30 does not mean that the disease is necessarily of short duration; five patients

10. Kunath, C. A.: The Stoneless Gallbladder, J. A. M. A. **109:** 183 (July 17) 1937. Smithies.[2]

11. Lahey, F. H., and Swinton, Neil: Stones in the Common and Hepatic Bile Ducts, New England J. Med. **213:** 1275 (Dec. 26) 1935.

12. Potter, A. H.: Gallbladder Disease in Young Subjects, Surg., Gynec. & Obst. **46:** 795 (June) 1928.

13. Blackford, J. H.; King, R. L., and Sherwood, K. K.: Cholecystitis: Study Based on Follow-Up After from Five to Fifteen Years of 200 Patients Not Operated On, J. A. M. A. **101:** 910 (Sept. 16) 1933.

(7.9 per cent) had symptoms for five or more years and one had symptoms for twelve years.

Only four patients (6.3 per cent) had acute cholecystitis and fifty-nine patients (93.6 per cent) had chronic cholecystitis. Gallstones were present in forty-one cases (65 per cent); this included three of acute cholecystitis.

Three patients had stones in the common duct, an incidence of 4.7 per cent, but when the incidence was based on the forty-one cases of calculous gallbladder it was 7.3 per cent. The duration of the symptoms in these cases varied from four to seven years.

Fifty-seven patients (90.4 per cent) were followed after operation; fifty-three (92.9 per cent) were free from symptoms when last seen. Thirty-six patients (97.2 per cent) of thirty-seven with cholelithiasis were entirely relieved; seventeen of twenty with noncalculous chronic cholecystitis (85 per cent) were either greatly improved or completely relieved.

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TREATMENT OF VULVOVAGINITIS WITH ESTROGEN

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Infections of the immature vagina present a social and medical problem of considerable magnitude, especially in hospitals and asylums for children. Despite the available diagnostic procedures, it is often most difficult to differentiate the transmissible (gonococcic) from the nonspecific form of vulvovaginitis. It therefore becomes imperative to isolate at considerable cost and inconvenience every institutional case of vulvovaginitis. The occurrence of gonococcic vulvovaginitis in the home of a family presents no less a problem in preventing the spread of the infection to young sisters and schoolmates. In many instances the condition remains unrecognized until two or more members of a family are affected.

Although serious complications such as arthritis, salpingitis and ophthalmia are relatively infrequent, unless the gonococcus assumes an unusual virulence in passing through one or more susceptible hosts even the mild form of the infection leaves its imprint on the psyche of the affected child. The persistence of vulvar irritation for many months or years centers the attention of the growing child on the generative organs and often leads to the habit of masturbation and premature heterosexual inclinations.

The incidence of gonococcic vulvovaginitis varies with the facilities of the community to detect and isolate infected children lacking proper supervision. Taussig [1] found that 5 per cent of the girls attending the dispensary of the St. Louis Children's Hospital had gonococcic vaginitis and that in most instances the parents or guardians were unaware of its existence. Compulsory reporting of cases to health authorities, education of parents in prophylaxis and better nursing facilities in hospitals have tended in recent years to decrease the incidence of the infection in most communities.

From the Philadelphia Institute for Medical Research in the Philadelphia General Hospital.

1. Taussig, F. J.: Diseases of the Vulva, New York, D. Appleton & Co., 1931.

ETIOLOGY OF VULVOVAGINITIS

Although the poor resistance of the ill child, as seen in the exanthems and in severe infections of the upper respiratory tract, plays an important role in the predisposition to vulvovaginitis, the most important predisposing factor is undoubtedly the low resistance of the immature vagina to any pathogenic bacteria, especially the gonococcus, which thrives in the alkaline medium occasioned by the glycogen-poor vaginal epithelium of the child.

The immature vagina, though not a sterile cavity, is zealously guarded against gross contamination and traumatism by the hymen until sexual maturity. Often the inexperienced nurse or intern, in making the routine vaginal smear preliminary to admission to the hospital, inadvertently brings the cotton swab into contact with the unclean external parts before the vagina is entered. This contamination plus the unavoidable friction-traumatism to the delicate vaginal epithelium frequently results in a nonspecific vaginitis, rendering the vagina more susceptible to invasion by the gonococcus. Such errors may be obviated, as suggested elsewhere by one of us,[2] by examining the centrifuged vaginal washings obtained through a small, soft rubber catheter after the vulva is thoroughly cleansed.

In institutional epidemics of gonococcic vulvovaginitis the disease is most often transferred through the medium of diapers, bed linens, rectal thermometers or the hands of careless nurses. The nursing technic in a pediatric ward should be based always on the assumption that one of the patients has an unrecognized gonococcic vulvovaginitis. In most instances of vulvovaginitis occurring in the home of the patient, the source of infection may be traced to an infected member of the family or to a contaminated lavatory seat at home or in school. According to Taussig,[1] this is the most frequent mode of transmission of the disease because of the characteristic manner in which children slide from high toilet seats.

DIAGNOSIS OF VULVOVAGINITIS

The clinical picture of vulvovaginitis is unmistakable. To differentiate the transmissible (gonococcic) from other types of vulvovaginitis requires careful bacteriologic study by smear and culture. The demonstration of gram-negative, intracellular diplococci, morphologically typical of gonococci, is fairly reliable. Unfortunately the smear method has its limitations and, when negative or doubtful, the more refined culture method is essential to a differential diagnosis. The relative merits of the two diagnostic procedures in children presenting definite clinical symptoms of gonococcic vulvovaginitis were evaluated by Ruys [3] as follows: "Smears positive, culture positive in 111; smears negative, culture positive in fourteen; smears positive or doubtful, culture negative in seven." The superiority of the culture method was thus amply demonstrated. Since Micrococcus catarrhalis, frequently present in the vagina of children, morphologically resembles the gonococcus, the culture method is especially valuable in instances of positive smears without clinical symptoms. One must nevertheless remember that even the culture method is not infallible, since an overgrowth of secondary invaders may obscure the picture. Negative cultures under these conditions do

2. Mazer, Charles, and Rubin, S.: Vulvovaginitis in Children, Am. Physician **31**: 653 (Dec.) 1926.

3. Ruys, A. Charlotte: The Etiology of Vulvovaginitis Infantum. J. A. M. A. **105**: 862 (Sept. 14) 1935.

not preclude the possible presence of gonococci. The gonococcus complement fixation test, even with the improved technic employed by Mascall,[4] is positive in only 58.6 per cent of cases of gonococcic vulvovaginitis. It is therefore safer to consider every case of vulvovaginitis as gonococcic when discovered in a pediatric ward or an asylum for children.

PATHOLOGY OF GONOCOCCIC VULVOVAGINITIS

In the acute stage the vulva is reddened and edematous. When viewed through an endoscope the vaginal mucosa appears congested and the site of small erosions. The portio vaginalis in the vicinity of the pinhead sized external cervical os presents the same pathologic picture, giving the erroneous visual and bacteriologic impression of an associated endocervicitis. We agree with Schauffler and Kuhn [5] that the endocervix of the child is practically never invaded by the gonococcus because of the rudimentary state of its racemose glands. This also explains the singularly low incidence of salpingitis in children affected with gonococcic vaginitis. None of the 118 patients in this series showed clinical evidence of salpingitis, nor did one of us (Mazer) observe a single instance of this complication during the severest institutional epidemic of gonococcic vaginitis and arthritis in medical history, which occurred in one of the Philadelphia maternity hospitals in 1924.[6]

Bartholinitis is extremely rare. It was not encountered in any of our 118 patients. The urethral meatus is often red and pouting, but Skene's glands are rarely infected. However, the presence of pus cells in the urine voided following cleansing of the vulva points to an anterior urethritis, which was encountered by Miller [7] in six of sixty-eight cases of gonococcic vulvovaginitis. Only one of the 118 cases herein reported presented a definite urethritis.

Proctitis is apparently a frequent complication and may be a source of vaginal reinfection when untreated. The smear method is evidently inadequate to demonstrate involvement of the rectum. By means of a special culture medium, Ruys [8] recovered gonococci from the rectum in every child afflicted with specific vulvovaginitis. Clinical evidence of an associated proctitis is, however, rarely seen except during an institutional epidemic of the disease, when, as shown by Cooperman,[6] even the male infants show symptoms of proctitis.

Arthritis is rarely encountered except during epidemics, when the gonococcus assumes an unusual virulence in passing through many nonresistant hosts. Illustrative of this was the high incidence of gonococcic arthritis encountered during the Philadelphia epidemic, in which twenty-seven males and forty females were equally affected with gonococcic arthritis.[6]

BASIS OF TREATMENT OF VULVOVAGINITIS WITH ESTROGEN

Owing to the presence of maternal estrogen, the vaginal epithelium of the newborn is of considerable thickness (from thirty to forty layers of cells). The numerous basal layers are compact and irregular, forming papillae which penetrate deeply the tunica propria. Superimposed on the basal epithelium are many layers of spindle-shaped, glycogen-containing cells. The vaginal secretion is acid and the bacterial flora predominantly of the bacillary type.[9] Thus the safeguards present at birth are similar to those current in the mature vagina, which is so highly resistant to infections other than Trichomonas vaginalis. Within three or four weeks after birth, when the influence of maternal estrogen subsides, the numerous layers of glycogen-containing epithelial cells are thrown off and the basal zone becomes thin and regular in outline—evidence of inactivity. There is a paucity in cell glycogen and of Döderlein's bacilli, resulting in alkalinity of the vaginal secretions.[10] The vagina remains in this vulnerable state throughout childhood until the advent of puberty, when the estrogen elaborated by the maturing ovaries causes a return of the vaginal mucosa to a neonatal-like state, varying only slightly in structure during the cyclic changes in production of estrogen by the active ovaries. With the advent of senility, the vaginal epithelium once again reverts to that resembling immaturity, becoming even more vulnerable to infection and traumatism than the childhood vaginal epithelium. The superficial layers disappear, leaving only about six layers of small nucleated cells with a granular cytoplasm.[11]

Knowledge of these facts led Lewis [12] to employ injections of estrogenic substances in the treatment of gonococcic vulvovaginitis in the hope of transforming the vulnerable childhood vagina to the resistant adult types. The new treatment suggested by Lewis evoked considerable discussion in the medical literature, some favorable and some entirely unfavorable.

In employing a new product in the treatment of human ailments, its safety must be established first. The most valid objection to the use of estrogen in children is the possibility of permanent damage to the ovaries. This objection, based on the experimental observation [13] that prolonged treatment with estrogen produces regressive ovarian changes, is difficult to substantiate in human beings. Very few of the children treated with estrogen for gonococcic vaginitis have to date attained puberty. None have attained the age of childbearing, to permit determination of the effect of the hormone on germ plasm.

In defense of the use of estrogen, the following evidence of its safety is available:

1. Rats and monkeys given considerable quantities of estrogen for a relatively long period ultimately become estrous, mate and reproduce normally.[14]

2. The blood of the unborn infant is normally surcharged with estrogen to a degree (at least 1 mouse unit per cubic centimeter) far greater than that of children treated with maximum doses of the substance.

3. The few treated children who have already attained puberty have menstruated at the predicted age.[15]

4. Mascall, W. N.: Pathological Diagnosis of Female Gonorrhea, Lancet **2:** 233 (July 29) 1933.

5. Schauffler, G. C., and Kuhn, Clifford: Information Regarding Gonorrhea in the Immature Female, Am. J. Obst. & Gynec. **25:** 374 (March) 1933.

6. Cooperman, M. B.: Gonococcus Arthritis in Infancy: A Clinical Study of Forty-Four Cases, Am. J. Dis. Child. **33:** 932 (June) 1927.

7. Miller, J. R.: Two Years' Experience with Theelin Treatment of Gonorrheal Vaginitis, Am. J. Obst. & Gynec. **29:** 553 (April) 1935.

8. Ruys, A. Charlotte, and Jens, P. A.: Kulturelle Untersuchung des Rektalschleimes bei Kindern mit Vulvovaginitis gonorrhoica, München. med. Wchnschr. **80:** 846 (June 2) 1933.

9. Cruickshank, Robert, and Sharman, Albert: Biology of Vagina in Human Subject; Glycogen in Vaginal Epithelium and Its Relation to Ovarian Activity, J. Obst. & Gynaec., Brit. Emp. **41:** 190 (April) 1934.

10. Lewis, R. M., and Weinstein, Louis: The Production of Vaginal Acidity by Estrin, Surg., Gynec. & Obst. **63:** 640 (Nov.) 1936.

11. Davis, M. E.: The Treatment of Senile Vaginitis with Ovarian Follicular Hormone, Surg., Gynec. & Obst. **61:** 680 (Nov.) 1935.

12. Lewis, R. M.: Study of Effects of Theelin on Gonorrheal Vaginitis in Children, Am. J. Obst. & Gynec. **26:** 593 (Oct.) 1933.

13. Allen, Edgar: Sex and Internal Secretions, Baltimore, Williams & Wilkins Company, 1932.

14. Allen, Edgar, and Diddle, A. W.: Ovarian Follicular Hormone Effects on Ovaries, Am. J. Obst. & Gynec. **29:** 83 (Jan.) 1935.

15. Mazer, Charles, and Israel, S. L.: Studies on the Optimal Dosage of Estrogens: An Experimental and Clinical Evaluation, J. A. M. A. **108:** 163 (Jan. 16) 1937.

4. Regularly menstruating women who receive estrogen in doses of 10,000 rat units every fourth day for periods varying from two to four months menstruate normally after withdrawal of treatment and bear normal offspring.[16] The more recent employment of vaginal suppositories, requiring, as it does, relatively small doses of estrogen, has partly solved the problem of possible harmful effects.

Next in importance to the safety of treatment of specific vaginitis with estrogen is its comparative effectiveness. Does it accomplish a cure sooner than other methods of treatment? Is the cure permanent or temporary? Opinions of competent clinicians concerning this phase of the problem differ so widely that, for the present, one must rely solely on personal experience. Dosage, length of treatment, mode of administration, attention to extragenital foci of infection not influenced by the substance and the ability to eliminate extraneous sources of reinfection undoubtedly account for the discordant reports. Dosage and route of administration are indissolubly linked, since even a small fraction of the hypodermic dose of estrogen produces the desired hypertrophy of the vaginal mucosa when introduced directly into the vagina.[17] Employment of the small doses required for vaginal administration of the substance reduces the incidence of the undesirable, though temporary and harmless, effects, such as enlargement of the breasts and premature menstruation, often encountered with effective hypodermic doses. There are, nevertheless, definite objections to prolonged vaginal treatment of the child. In addition to requiring intelligent cooperation of the mother, who, despite her intelligence, may nevertheless inadvertently carry the infection to another member of the family, oft-repeated vaginal treatment of the child frequently creates the habit of masturbation and premature heterosexual inclinations.

The size of the dose is the determining factor in accomplishing a cure. The length of treatment determines the percentage of recurrences. The longer the period of treatment, the less the chance of recurrence. The dose must be sufficient to cornify the vaginal epithelium and to reduce the p_H of the vaginal secretions to a point below 6. Our experience has shown that the minimum effective hypodermic dose is 500 rat units given every other day. Smaller doses, such as 100 rat units (approximately 800 international units), even when administered daily, produce only doubtful vaginal reactions and require treatment for as long as twenty-two weeks.[18] The minimum dose by the vagina is 100 rat units nightly, inserted on retiring. With these doses, the necessary vaginal response (cornification, acidity and an absence of pus and gram-negative diplococci) is usually observed on the fourth week of treatment.

In considering the problem of dosage, it is pertinent to clarify the existing confusion which dual standardization of estrogenic products creates. Estrogenic substances vary widely in their ability to cornify the vaginal mucosa of the rat. The international unit (weight unit) of estrogen is 0.1 microgram. The rat unit (biologic unit) is the minimum quantity of estrogenic substance capable of evoking estrus in the castrated rat. The ratio between the international unit and the rat unit varies with the chemical composition and the purity of the product. In our laboratory, various samples of estrone (theelin) yielded 1 million rat units per gram, estriol (theelol) 150,000 rat units per gram, estradiol (progynon-DH) as much as 10 million rat units per gram, and estradiol benzoate (progynon-B) 5½ million rat units per gram. It is evident, therefore, that the use of the rat unit is preferable when dealing with the several estrogens.

Equal to the importance of dosage in accomplishing clinical and bacteriologic cure is the length of treatment in preventing recurrence of the infection. It will be seen later that the percentage of recurrences is larger when the period of treatment, despite early and repeated negative smears, is much less than eight weeks. We [15] have previously stressed the importance of the length of treatment in preventing recurrences. Lewis [18] is equally emphatic on this point. The truth of the dictum that too short a period of treatment and not reinfection is responsible for the majority of early recurrences is evident from a study of some of the available literature on hypodermic treatment of vulvovaginitis with estrogen. Regardless of dosage, which if inadequate could not have produced even a temporary cure, too short periods of treatment (from twenty-one to forty days) allowed recurrences in nine of twenty-two patients treated hypodermically by Te Linde,[19] in seven of thirteen treated by Phillips,[20] in four of eleven treated by Brown [21] and in eight of twenty-two children treated by Reichert.[22] On the other hand, with longer treatment (from fifty to sixty days), Limper [23] observed no recurrences in twenty-four patients discharged as cured, and Lewis [18] encountered none in twenty-two. Recurrences appearing soon after the vagina returns to its former immature state (four weeks after treatment) may be reasonably attributed to insufficient length of treatment and not to reinfection. Those appearing later than three months after withdrawal of treatment may be attributed to reinfection or to a persistent focus not within the influence of the substance.

EVALUATION OF TREATMENT WITH ESTROGEN IN 118 CASES OF GONORRHEAL VULVOVAGINITIS

The present study embraces a group of 118 children, ranging in age from 18 months to 11 years, admitted consecutively to the wards of the Philadelphia General Hospital from January 1935 to May 1937 for the treatment of gonococcic vulvovaginitis. The diagnosis in each case was based on clinical symptoms and on the presence of gram-negative, intracellular diplococci, structurally characteristic of the gonococcus in the vaginal smear. Since the children were hospitalized during the entire period of treatment, there was an excellent opportunity to observe the response to the administration of estrogen. About one third of the children were in the acute stage of the illness at the time of admission. The duration of the disease in the others ranged from two months to two years. There was

16. Mazer, Charles; Meranze, D. R., and Israel, S. L.: Evaluation of the Constitutional Effects of Large Doses of Estrogenic Principle, J. A. M. A. **105:** 257 (July 27) 1935.
17. Berger, Milan: Besonders hohe Wirksamkeit des Follikelhormons bei vaginaler Instillation, Klin. Wchnschr. **14:** 1601 (Nov. 9) 1935.
18. Lewis, R. M., and Adler, E. L.: Gonorrheal Vaginitis: Results of Treatment with Different Preparations and Amounts of Estrogenic Substance, J. A. M. A. **106:** 2054 (June 13) 1936.
19. Te Linde, R. W., and Brawner, J. N., Jr.: Experiences with Amniotin in the Treatment of Gonococcal Vaginitis in Children, Am. J. Obst. & Gynec. **30:** 512 (Oct.) 1935.
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no appreciable difference in the response of the respective groups to treatment with estrogen.

Eighty-one of the 118 children were treated by means of hypodermic injections of estradiol benzoate (progynon-B [24]); thirty-four received the same product as vaginal suppositories, and the remaining three were given estradiol (progynon-DH) orally.

HYPODERMIC ADMINISTRATION OF ESTROGEN

Sixty-one of the eighty-one children hypodermically treated received from 1,000 to 1,500 rat units and seven were given 500 rat units every other day for eight weeks; the remaining thirteen received 500 rat units every other day for a period of from four to six weeks.

The first of four consecutive vaginal smears was obtained after two weeks of treatment in forty-six cases, in the fourth week of treatment in fourteen and during the fifth week in eighteen. The vaginal discharge was then scanty, thick and snow white, revealing a mass of non-nucleated epithelial cells and gram-negative bacilli on smears. In the remaining three the vaginal smears were persistently positive, possibly because of inadequate vaginal drainage of accumulated débris, which in itself may have been a source of irritation. In order to evaluate treatment with estrogen accurately, no attempt was made to dilate the introitus or to irrigate the vagina for the purpose of removing the plugs of desquamated epithelial cells.

Sixty-one of the seventy-eight children successfully treated with estrogen hypodermically were followed for a period of from three to twenty-three months, with an average of ten months for the entire group. There were only six (10 per cent) recurrences in the sixty-one followed-up patients who had received hypodermic treatment. Analyzing the sixty-one followed-up cases according to the length of treatment, we found four recurrences in the fifty-three who had received treatment for eight weeks and two in the eight who had received treatment for a period of only from four to six weeks. Notwithstanding the fact that the comparison is made on too few patients, it is nevertheless significant that the incidence of recurrences in the group of eight followed-up patients who had received the relatively short treatment was three times greater than in the group treated for the full period of eight weeks. Equally significant is the fact that recurrences with shorter treatment were not only more numerous but appeared much earlier (from four to six weeks after treatment), suggesting that incomplete cure was the cause of the trouble.

Side effects other than growth of pubic hair occur more frequently and are more intense with adequate hypodermic treatment than with vaginal suppositories. Twenty-one of the eighty-one children (26 per cent) showed enlargement of the breasts, which persisted for about a month after withdrawal of the treatment; seven (8.5 per cent) had a scanty growth of pubic hair, which eventually disappeared, and eight (10 per cent) had a single episode of uterine bleeding either during treatment or later.

TREATMENT WITH VAGINAL SUPPOSITORIES OF ESTROGEN

Thirty-four children were treated with vaginal suppositories, each containing 200 rat units of estradiol benzoate (progynon-B), inserted nightly on retiring for a period of eight weeks.

A clinical cure and four consecutive negative vaginal smears were obtained during the fourth week of treatment in thirty-three of the thirty-four children. The remaining patient failed to respond to vaginal as well as to subsequent hypodermic treatment with 1,500 rat units given every other day for a period of eight additional weeks.

The side effects with vaginal suppositories of estrogen were less pronounced than with hypodermic treatment. In eight of the thirty-four patients there was a moderate enlargement of the breasts; in six there was a semblance of pubic hair growth and in one a single episode of menstruation. This child was, moreover, 11 years old and might have menstruated irrespective of the treatment.

Twenty-six of the thirty-three children treated with vaginal suppositories and discharged as cured were followed for a period of from three to thirteen months, averaging seven months. There were no recurrences, essentially substantiating the observation of Te Linde [19] that the treatment of gonococcic vulvovaginitis with vaginal suppositories is practically free from recurrences. He [25] states that a check-up of his first 100 patients treated by vaginal suppository showed ninety-eight of them clinically well and with negative smears from three months to two and one-half years after the last treatment.

ORAL TREATMENT

In three patients, oral administration of 1,500 rat units daily of estradiol (progynon-DH) for a period of eight weeks failed to accomplish a cure. They were subsequently treated by other methods. These results with oral therapy are in accord with those obtained by Te Linde [19] in six patients treated with equally large doses for a period averaging 105 days. It seems that in the human being as well as in the experimental animal [26] the minimum effective oral dose of estrogen must be five times greater than the hypodermic dose.

SUMMARY

1. The basis of treatment of vulvovaginitis with estrogen is the ability of the substance to create a temporary maturity environment in the vagina of the child and thus render it resistant to pathogenic bacteria.

2. The dose of estrogen must be sufficient to cornify the vaginal epithelium and to reduce the p_H of the vaginal secretions to a point below 6.

3. Treatment for eight weeks, despite an earlier clinical and bacteriologic cure, is a safeguard against recurrence of the infection.

4. Hypodermic injections produced a clinical and bacteriologic cure in seventy-eight of eighty-one children, with a 10 per cent incidence of recurrence.

5. Treatment with vaginal suppositories of estrogen produced a clinical and bacteriologic cure in thirty-three of thirty-four children without any recurrence in the twenty-six who were observed for a relatively long time.

6. Oral treatment was ineffective in three children who received as much as 1,500 rat units daily for a period of eight weeks.

7. Side effects, such as growth of pubic hair, uterine bleeding and enlargement of the breasts, are temporary and are more frequently encountered with hypodermic than with local treatment with estrogen.

2047 Spruce Street.

24. Drs. Gregory Stragnell and Erwin Schwenk supplied the material employed in this work.

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LOCAL AND SYSTEMIC EFFECTS FROM INHALATION OF STRONG SOLUTIONS OF EPINEPHRINE

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Inhalation of vaporized 1 per cent solution of epinephrine is extensively used in the relief of bronchial asthma.[1] This is a convenient way of obtaining directly the well known bronchodilator action of epinephrine without the necessity of hypodermic injection. The clinical results have been generally good as to relief of asthmatic attacks. At least ten atomizers are advertized as specially designed for this purpose, and, in addition, 1 per cent solution of epinephrine is the active ingredient of a large number of trade-marked antiasthma products offered to the public. In spite of the widespread use of this strong concentration of epinephrine, no reports have been made as to possible local irritative effects on the respiratory tract. When it is remembered that deep inhalations of this potent drug are practiced almost indefinitely in a chronic disease state, it would appear desirable to understand more fully and definitely, if possible, the mechanism of the antiasthmatic action and the possible side actions locally and systemically. Therefore, studies were initiated toward these ends and are reported in this paper.

SIZE OF DOSE

The vaporizers ordinarily used are specially designed to throw out very fine impalpable droplets, which are so small that they will not wet or fog a mirror. In order to determine how much of the solution was vaporized under conditions of ordinary use, three hand atomizers, or nebulizers, of the type prescribed for epinephrine inhalations were weighed on an analytic balance before and after use on ten different occasions. After squeezing the bulb forty times, which is more than is commonly done by patients, there was delivered an average of 0.034 Gm. of the 1 per cent solution, or 0.34 mg. of epinephrine from the mouth of the nebulizer, or about 0.008 mg. of epinephrine per compression. Only a small portion of this solution reaches the bronchi when the vapor is inhaled through the mouth. Spoto [2] working with dogs and Rubow [3] with rabbits estimated the penetration of such nebulized fluids into the deep respiratory passages by using solutions of chemicals which could be quantitatively determined in the lung tissue. They found that not more than 3 per cent of inhaled vapor reached the lung, and generally the amounts were much less. According to these authors the greater part of the solution was deposited on the pharynx, larynx and trachea.

Similar localization was confirmed for patients by the following observation:[4] A surgical patient, with a bronchial fistula in the anterior wall of the chest communicating with the left main bronchus, was prepared for excision of the fistula. A 5 per cent aqueous solution of gentian violet was continuously vaporized into the patient's mouth for several minutes while he inhaled deeply and exhaled through the fistula. A small catheter in the fistula conducted the expired air through a test tube of water, but this remained uncolored. The operation then proceeded, with excision of the fistulous tract. Examination of it immediately after removal failed to reveal the slighest trace of staining of the tissue by the gentian violet, although a blue color was present in

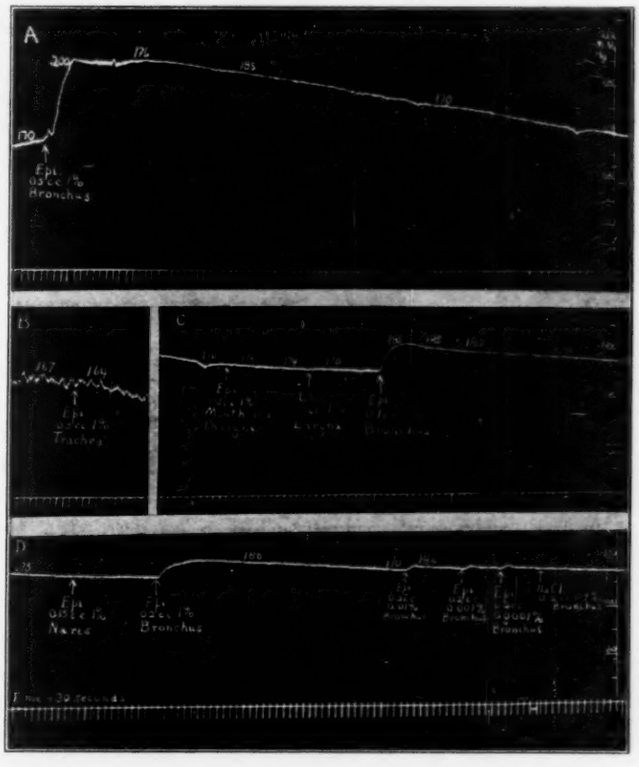

Fig. 1.—Effects on the blood pressure of a cat from different volumes and strengths of solution of epinephrine applied to isolated segments of the respiratory tract (pentobarbital anesthesia).

the mouth and posterior portion of the pharynx. Obviously not enough gentian violet reached the bronchus to impart a color to the water or to stain the walls of the fistula. From the fact that the tinctorial capacity of aqueous solutions of gentian violet is very great, a definite color being present in dilutions as low as 1:10,000,000, the total amount of the dye in the air expired must have been negligible. The mechanisms for removing particulate matter from the inspired air must be extremely efficient, as practically all the droplets were removed from the air before it reached the smaller bronchi. This agrees with Spoto's and Rubow's observations that the greater part of a vaporized solution remains in the upper part of the respiratory passages. In view of this result, it was of interest to determine whether the epinephrine was absorbed from the upper part of the respiratory tract and exerted any systemic effects.

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CIRCULATORY EFFECTS

To this end the pressor and cardiac effects of inhalation of epinephrine in twelve asthmatic patients and four normal persons were determined as a measure of systemic absorption. The patients were all accustomed to the use of the nebulizer and were tested during an acute asthmatic attack. Pulse rates and blood pressures

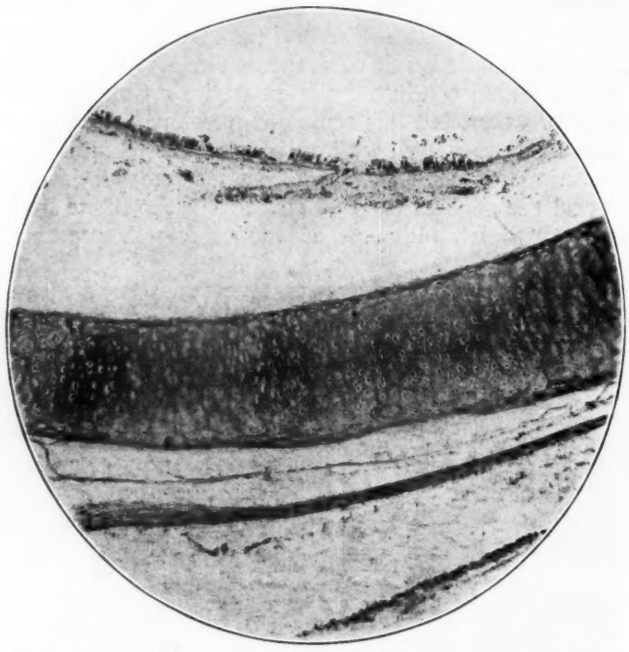

Fig. 2.—Microscopic section of trachea of cat after inhalation of 1 per cent solution of epinephrine, showing loss of cilia and desquamation of epithelium, together with beginning leukocytic infiltration of submucosa.

were determined every two minutes during ten minute periods before and during inhalation of the vapor and for ten minutes afterward. The administration consisted of ten deep inspirations with the nebulizer held just within the mouth. No changes in pulse rate or blood pressure were observed in either of the groups tested. Two of the normal persons noted subjective signs of palpitation and vertigo, but they were able to produce the same sensations by deep inhalations alone without epinephrine. These results are in general agreement with the clinical reports [1] and indicate that a systemic action of epinephrine under the conditions is not common.

Animal experiments were next made for more precise determinations of systemic effects under better controlled and more varied conditions. Cats anesthetized with intraperitoneal injection of pentobarbital sodium, 35 mg. per kilogram of body weight, were used. After tracheotomy, a section of the trachea was isolated by ligation from the larynx above and the bifurcation below in order to divide the respiratory tract into three parts, namely mouth and nasopharynx, trachea, and pulmonary or bronchial airways. Arterial blood pressure was measured by means of a cannula inserted into a common carotid artery and connected to a mercury manometer recording on a kymograph. The pulse rate was counted every five minutes or oftener.

The results of instilling different volumes of solution of epinephrine into these three regions are illustrated in figure 1. No change in pulse rate or blood pressure was recorded after instillation of such large doses as 5 mg. of epinephrine into the mouth, nasopharynx or isolated trachea. That is, typical systemic effects were not demonstrated after application to the mucosa above the bifurcation of the trachea. The bronchial mucosa, however, proved to be a good absorbing surface for epinephrine, since pulse and blood pressure changes were demonstrated after as little as 0.0002 mg. deposited in a bronchus. Considering the differences in body weight and the amounts lost in the upper air passages, a patient would require almost 100 inhalations to get an equivalent amount into his lungs and therefore to get systemic effects. This quantity could be obtained more readily, however, if compressed air were used for the vaporization, as is sometimes practiced in physicians' offices.

The failure to produce any effects from the excessive quantities applied to the tissue in the upper part of the respiratory tract makes it improbable that the solution deposited in this region is effective, acting either by reflex irritation or after absorption. In addition, the previous demonstration that to get systemic actions from the solution deposited deep in the lungs would require a much more prolonged period of inhalations than is ordinarily used for therapeutic effects makes it improbable that the effectiveness depends on getting enough absorbed in the lungs for a systemic action. Therefore the anti-asthmatic actions are probably the result of the direct local effects of the epinephrine on the congested bronchial mucosa and spastic musculature, occurring independently of absorption into the general circulation.

EFFECTS OF CHRONIC INHALATION

Repeated inhalations of this strong solution of epinephrine might be expected to cause local irritation and damage to the mucosa of the upper airways, since more

Fig. 3.—Microscopic section of trachea of cat after inhalation of 1 per cent solution of epinephrine. Ulceration of mucosa with loss of epithelium, marked leukocytic infiltration of submucosa and lumen filled with mucopus.

than 95 per cent of the inhaled solution is deposited in this region. Therefore ten cats and six rabbits were given the vapor for a determination of the local effects on the tissues. They were healthy animals, maintained on the standard diet of the laboratory colony. The animals were treated as follows: 1. Four rabbits and six cats were given inhalations of 1 per cent solution of epinephrine twice a day six days a week for a maxi-

mum of 121 days for the rabbits and seventy-one days for the cats. The dose was ten squeezes of the atomizer bulb or 0.0085 cc. of the solution of epinephrine delivered into the mouth as the vapor at each administration. 2. As controls, two cats and two rabbits inhaled at the same time an equal volume of physiologic solution of sodium chloride vaporized with a similar nebulizer. All inhalations were given through a mouth gag with the special nebulizer recommended for patients, and the jets of vapor were synchronized with the animals' inspiration in the same manner as is practiced by patients. 3. Because Graeser and Rowe and others [1] have reported gastrointestinal upsets in patients using epinephrine by inhalation, which they ascribed to swallowing the larger droplets, two cats were made to drink 1 cc. of 1 per cent solution of epinephrine six times each week. This was continued for twenty-two days with one cat; the other cat died on the seventh day. Histologic sections were made from the respiratory tracts of all the animals dying during the course of the experiment or killed at the conclusion of the medication. The accompanying table summarizes the histologic changes observed.

The most frequent abnormality was the presence of mucopus in the lumens of the bronchi. This occurred in seven of the ten animals receiving inhalations of epinephrine and in three of the six controls. The significance of this exudation is doubtful. More significant was the change illustrated in figure 2 of loss of tracheal cilia in six of the medicated animals and desquamation of tracheal and bronchial epithelium in four. These changes were not observed in any of the controls. In three of the epinephrine-sprayed animals there was a leukocytic infiltration of the tracheal submucosa which was sufficiently generalized to be termed a tracheitis, as shown in figure 3. Two cats from this group also showed evidence of bronchitis. These inflammatory reactions were not seen in the unmedicated controls. Five of the ten animals taking inhalations of epinephrine died during the course of the experiments after an average of thirty-seven days, the animals that remained being killed after an average of eighty-seven days.

Both of the animals receiving solution of epinephrine by mouth showed evidence of severe diarrhea, marked anorexia and loss of body weight, as did also one of the cats receiving the inhalations of epinephrine. Postmortem examination of the intestinal serosa and peritoneum of these three cats indicated the presence of inflammation, and the gastrointestinal mucosa showed many small ulcerated and hemorrhagic areas. Accordingly, these applications of epinephrine produced the typical signs of local irritation in both pulmonary and gastrointestinal tissues.

The question might be raised whether similar effects might be produced in patients. It is obvious that the opportunity of examining the trachea and lungs of patients receiving inhalations of epinephrine for asthma would be comparatively rare. However, we had an opportunity of examining one patient with conditions which were confirmatory of the changes in the cats. The pertinent facts of this case may be presented, with the histologic pictures, as follows:

A man aged 60 with advanced pulmonary tuberculosis showed no clinical signs of tracheal or laryngeal involvement. Inhalations of 1 per cent solution of epinephrine were given every two hours for forty-eight hours before death. At autopsy, which was performed immediately, the trachea was reddened down to within a short distance above the bifurcation. Microscopic examination revealed marked leukocytic infiltration throughout the reddened area (fig. 4) and areas of complete loss of epithelium with leukocytic infiltration of submucosa (fig. 5). Special stains revealed no evidence of tuberculous processes in this region, and the larynx and the bronchi below the bifurcation were free of these acute inflammatory changes, or tuberculosis. The pathologic diagnosis at this part of the autopsy was acute tracheitis produced by the inhalations.

COMMENT

The amount of epinephrine obtained from a 1 per cent solution by proper vaporization for inhalation is considerably less than the usual dose with other methods of administration. A patient taking ten deep inhalations of the vapor from a hand atomizer receives not more than 0.08 mg. of epinephrine into his mouth. Only about one one-hundredth of this quantity gets down to the bronchioles, where it produces its therapeutic effects. The minimum intravenous dose of epinephrine required to produce just demonstrable systemic effects is of the order of 0.001 mg. per kilogram of body weight, or about 0.07 mg. for an average adult. Assuming that the total amount of vapor sprayed into the mouth reached the lung and was instantly and completely absorbed, the dose of epinephrine would be barely enough to produce a trace of action after systemic distribution. However, since not more than about one one-hundredth of the inhaled epinephrine vaporized

Pathologic Changes in Rabbits and Cats Given 1 per Cent Epinephrine or Physiologic Solution of Sodium Chloride Twice a Day by Inhalation or 1 per Cent Epinephrine by Mouth

Animal and Number	Fate	Bronchopneumonia	Bronchitis	Tracheitis	Mucopus in Lumen	Desquamation of Epithelium	Loss of Cilia	Comment
Epinephrine 1 per Cent Solution by Inhalation								
Rabbit 1	Died 34 days	..	..	..	..	..	..	No gross or microscopic pathologic changes
Rabbit 4	Died 117 days	+	..	..	+	+	+	Pleural empyema
Rabbit 3	Killed 121 days	..	..	..	..	..	+	Pathologic changes limited to trachea
Rabbit 2	Killed 121 days	..	..	+	+	+	+	Pathologic changes limited to trachea
Cat 3	Died 6 days	..	..	..	..	..	..	Normal respiratory tract; marked gastro-enteritis
Cat 1	Died 8 days	+	+	+	+	+	+	Extensive inflammatory changes
Cat 2	Died 18 days	+	+	+	+	+	+	Lower half of trachea plugged with mucus
Cat 6	Killed 54 days	..	..	..	+	..	..	
Cat 4	Killed 71 days	..	..	..	+	..	..	No changes in lungs or small bronchi
Cat 5	Killed 71 days	..	..	..	+	..	..	
Physiologic Solution of Sodium Chloride Inhalation								
Rabbit 1c	Died 82 days	+	+	..	..	..	..	
Rabbit 2c	Killed 121 days	..	..	..	..	..	..	Normal
Cat 1c	Killed 71 days	..	..	..	+	..	..	Trachea and lungs normal
Cat 2c	Killed 71 days	..	..	..	+	..	..	Trachea and lungs normal
Epinephrine 1 per Cent Solution by Mouth								
Cat 11	Died 7 days	..	..	+	+	+	..	Gastro-enteritis and loss of weight
Cat 12	Killed 22 days	..	..	..	..	..	..	Gastro-enteritis and loss of weight; respiratory tract normal

in the mouth gets to the lungs, it is improbable that enough would be absorbed for a circulatory action. Therefore it is not surprising that clinical pulse rate or blood pressure changes are usually not demonstrable after inhalation of epinephrine. Nielsen reached the same conclusion when he found the dose of epinephrine

Fig. 4.—Microscopic section of tracheal mucosa of a patient who received inhalations of 1 per cent solution of epinephrine every two hours for forty-eight hours preceding death; marked leukocytic infiltration.

by inhalation to be considerably less than the systemic dose for an effect in the same patient. These results taken together indicate that the relief of asthmatic symptoms after inhalations of epinephrine is the result of a local or direct action of the epinephrine on the bronchial muscle or mucosa, the greater effectiveness for this condition being presumably due to the direct contact of the drug with the site of action.

As for the possible objectionable side actions from repeated inhalations of epinephrine, very definite pathologic changes were demonstrated by us in the respiratory tissues of some of the animals used and in the patient who had inhaled 1 per cent solution of epinephrine. In spite of the small total amount of epinephrine that is deposited on the mucosa, each droplet contains a very high concentration of the drug. It may be that the local irritant effect of each droplet is just as great for the point of deposition as that which would result from the application of a greater volume to a proportionately larger area.

The type of lesion observed in the tracheal and bronchial mucosae is in many ways similar to lesions produced by Fox [5] by spraying the nasal mucosa of rabbits with 1:1,000 solution of epinephrine hydrochloride. After daily applications for three months he found considerable denudation and intra-epithelial abscesses with leukocytic infiltration. The irritant power of even this ten times weaker solution is therefore well known for the nasal mucosa in animals as well as in patients. There can then be little doubt that a stronger solution, i. e. ten times, or the 1 per cent strength, would produce greater local irritation, as was indeed demonstrated by us.

The solution of epinephrine used in our experiments, like many epinephrine products on the market, contains about 0.2 per cent sodium bisulfite to prevent oxidation of the drug. It has not been determined whether the irritation in the respiratory tract is due to the epinephrine itself, to the high acidity or to the preservative. In a recent publication, Tainter, Throndson and Lehman [6] described the irritant actions of sodium bisulfite on other mucous membranes. They showed that concentrations of this salt comparable to those in commercial epinephrine products were sufficient to produce local inflammatory reactions. However, the vasoconstriction probably contributes mainly to the local reaction, since it is well known that concentrations of epinephrine greater than 1:10,000 are apt to produce such prolonged vasoconstriction as to cause tissue damage from anoxemia with secondary inflammatory reactions.

Finally, there may be considered the bearing of these various observations on the clinical use of inhalations of epinephrine. There is no doubt that in many patients asthmatic attacks can be controlled by inhalation of strong solutions of epinephrine. There can also be little doubt that in some if not all patients the solution of epinephrine will produce inflammatory changes in the upper respiratory passages and possibly also in the gastrointestinal tract if droplets are swallowed. The more efficiently the patient breathes and draws the vapors into the deeper airways, the more extensive will be the inflammatory changes. If the concentration of the epinephrine should be increased from the 1 per cent strength now commonly employed to the 10 per cent strength advocated by Nielsen,[1] the possibility of local

Fig. 5.—Another area from same patient, showing complete loss of epithelium and more marked round cell infiltration.

injurious effects would be still greater. The main reason for using the inhalation procedure is that it saves the patient the inconvenience of hypodermic injections. However, if this advantage is obtained at the expense of setting up inflammatory reactions in the mucosae of the respiratory passages, which are already the seat of a morbid process, the secondary effects of the inhalation

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may prevent or delay restoration of these tissues to normal, even though the spasmodic attack is alleviated. The clinician would therefore seem to be under the necessity of deciding in each individual case whether the gain in convenience of medication by inhalation is great enough to justify the possible production of added pathologic changes.

SUMMARY AND CONCLUSIONS

1. Epinephrine, in strengths of 1 per cent or more, is used extensively by inhalation in the treatment of asthma. This procedure introduces smaller quantities of the drug into the lung than those clinically effective by injection.

2. There is no demonstrable absorption of epinephrine from the respiratory tract in cats, except deep in the lungs. However, from the lungs, blood pressure effects can be produced from minute doses, indicating good absorption from the lower respiratory mucosa, and the possibility of producing direct effects by the drug on the bronchiolar muscle and mucosa.

3. Inhalation of 1 per cent solution of epinephrine at frequent intervals in cats and rabbits produces in some animals an irritation of the tracheal mucosa, with desquamation of the epithelium and inflammatory changes. Similar changes were observed in the trachea of a tuberculous patient similarly medicated for forty-eight hours before death.

4. Careful consideration should be given by the physician to the possible occurrence of undesirable local irritative effects from inhalations of epinephrine such as those demonstrated by us, and whether these are great enough in the individual patient to outweigh the convenience of taking the drug by inhalation rather than by injection.

Sacramento and Webster streets.

Clinical Notes, Suggestions and New Instruments

ADENOCARCINOMA OF THE BREAST COINCIDENTAL WITH STRENUOUS ENDOCRINE THERAPY

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Estrogenic substance induces mammary cancer in mice usually only after several months' application. The carcinogenic agents likewise require considerable periods of application. Injections of estrogenic substance or of carcinogenic chemicals in rather excessive doses may shorten the time of appearance of experimental tumors.

The ordinary therapeutic doses of estrogenic substance and the rather short periods of treatment should not involve a risk of the subsequent development of cancer. A substitution therapy extending over many months does involve such a risk, especially if the woman treated presents a family history of mammary cancer and may thus supposedly be classed as susceptible.

Cramer [1] has presented an excellent review of the etiology of mammary cancer in man and in mice. Olch [2] expressed the belief that prolonged ovarian activity during the usual menopausal ages is detrimental or of no benefit to the mammary parenchyma. Crossen and Hobbs [3] noted the high incidence of delayed menopause in cases of adenocarcinoma of the body of the uterus. Wassink's [4] statistical study on Dutch people showed that family history, as regards mammary cancer, was exceedingly important. Waaler's [5] observations in Norway lead to similar opinions. Lacassagne [6] suggested that in experimental animals the hereditary factor for cancer of the breast may consist in an unequal response of the breast in the different strains to similar doses of estrogenic substances. Cramer [7] more recently demonstrated that mouse strains bearing a high incidence of cancer of the breast are more susceptible to estrogenic substance. It appears also from his work that the pituitary thyrotropic factor antagonizes the susceptibility to estrogen. A clinical investigation to determine if possible methods of identifying women who are unusually responsive to estrogenic agents is urgently needed. There is also much room for consideration of the family histories in the early diagnosis of mammary cancer.

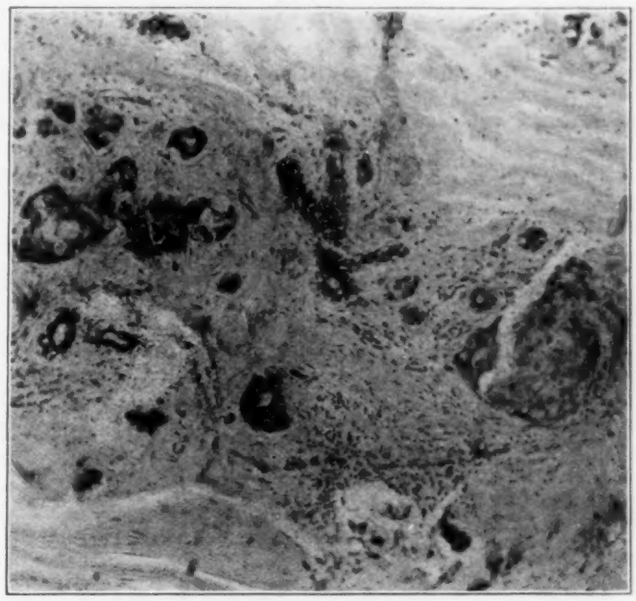

Fig. 1.—Section of the original tumor, showing obliteration of the ducts and fibrosis.

The following case is offered as an instance of what we believe to be an unwise use of estrogenic substance during the menopausal period. The adenocarcinoma of the breast in this instance, coincidental though it may be, tends to incriminate the heavy and prolonged estrogen treatment used:

REPORT OF CASE

History.—A white woman aged 50, a nurse, never married, entered the hospital in February 1938, complaining of a tumor mass in the right mammary gland, slightly painful and tender to pressure. There had been no loss of weight. The onset had ocurred three weeks before admission to the hospital. While reaching to clean a wall of her home she had felt a pain in her right breast, and on examination she observed a lump which was tender to palpation. On consulting a physician she was referred to the hospital.

Menstruation began at the age of 11 years and continued regularly to the age of 48. In September 1936 she consulted a physician for symptoms of weakness and fatigue following

From the Tumor Clinic and Cancer Research Unit, Veterans Administration Facility.

Drs. Hugh Scott, W. E. Kendall and L. H. Prince and Mr. R. P. Creer assisted in the preparation of this paper.

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physical exertion. She was told that her blood pressure was low and was given estrogenic therapy, receiving 2,000 units intramuscularly twice a week for twenty-six weeks. At this time she consulted another physician, and this type of therapy was continued. She was given one injection of 10,000 units of estrogen and then 2,000 units three times a week for six months. During this time she did not menstruate, but occasional fulness of the breast was noted. In the fall of 1937, when she voluntarily stopped the injections, there was a copious menstrual flow lasting one week. There has been none since.

She had had measles, mumps and chickenpox in childhood and pneumonia at the age of 9 years and again at 20, with complete recovery. Tonsillectomy had been done in 1920. She had been admitted because of a mild depressive psychosis in March 1931 and discharged improved in July 1931. She was readmitted in October 1935, when an atypical mucous cyst of the lower lip was removed and hyperkeratosis of the left cheek was treated by superficial irradiation. She was discharged in November 1935, with maximum benefit. She had always been declared competent as to her psychosis, and at this time, although worried about her breast, she had good insight and judgment was not defective.

Her mother died at the age of 63 of carcinoma of the liver and her father at 72 of nephritis and cardiac disease. Five

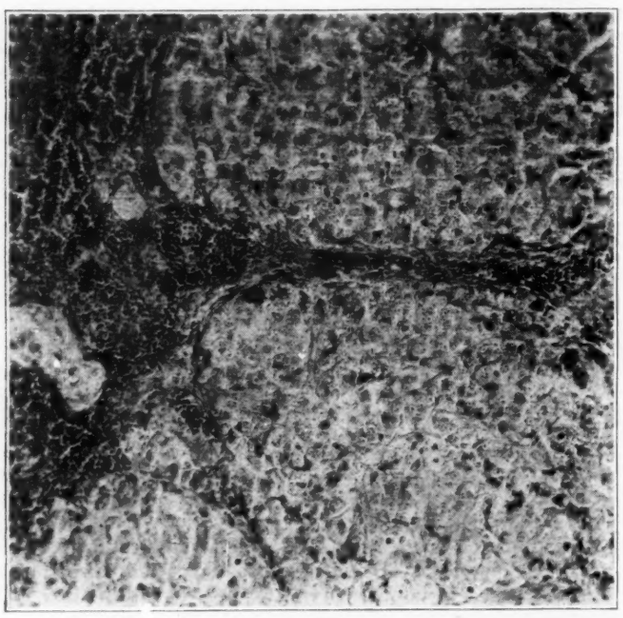

Fig. 2.—Section of metastatic nodule.

brothers reached adulthood but had all died from various causes other than cancer. Four older married sisters had no history of cancer.

Examination.—The patient was well developed and somewhat obese. Her height was 67 inches (170 cm.) and her weight 186 pounds (84.4 Kg.). In the upper outer quadrant of the right breast a tumor mass 5 by 7 cm. in diameter was present. This was not attached to the deeper structures or to overlying skin. There was no retraction and no discharge from the nipple. No adenopathy was noted in the drainage areas. The left breast was apparently normal. General examination showed no further abnormality. The urine showed a trace of albumin and a moderate number of white blood cells. The Wassermann and the Kahn test were negative. A blood count was within normal limits. X-ray examination of the chest showed no abnormality.

Course.—Radical mastectomy was performed, with careful dissection of the axilla and removal of both pectoral muscles. A study of the removed specimen showed several small nodes just adjacent to the primary tumor. Microscopic examination showed adenocarcinoma in both the primary tumor and the metastatic nodes.

The patient received a full course of postoperative irradiation.

COMMENT

The total dose of estrogen (258,000 units injected) used in this case may not seem high. The therapy, however, was practically continuous for over a year. That no menstrual periods occurred in this interval rather substantiates the factor of continuous dosage, as do the prolonged menses when the treatment was discontinued. The patient's history indicates periodic if not rather constant stimulation of the breast during the period of therapy. This feeling of fulness in the breast was not noted after discontinuance of the estrogen therapy. There was but one instance of cancer in the family and that not cancer of the breast.

In our experience papillomas of the mammary duct similar to that noted in Schimmelbusch's disease may be induced in experimental animals in short periods with low doses of estrogen. More prolonged experimental treatment brings forth typical tumors of the breast. It is not our intent to generate cancerphobia or to discourage the correct therapeutic use of estrogenic preparations. We feel, however, that there is some question whether prolonged and continuous therapy, as in the case reported, is compatible with the normal physiologic endocrine balances. At the menopause nature is attempting to close a period and to readjust biologically the tissues concerned. Correct endocrine therapy should, it would seem, be planned to aid the natural physiologic procedures. Continuous estrogen therapy would maintain this agent at a high level in the body. A consistently high estrogen level is certainly unphysiologic as is evident from studies on estrogen excretion. More attention should be given to timing and to tapering off the doses of estrogen when this agent is employed for therapeutic effect during the menopausal period.

ANTERIOR POLIOMYELITIS TREATED WITH SULFAPYRIDINE

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In view of the variation in the severity of the infection causing anterior poliomyelitis, with its differences in clinical picture, its symptomatology and its resulting paralysis, one case report is not of great value. The case here reported is interesting because the patient was seen during the acute stage and rather surprising results were had accompanying the use of sulfapyridine in the treatment.

History.—M. E. M., a well developed girl aged 9 years, weighing 65 pounds (29.5 Kg.), was first seen March 22, 1939, complaining of pain in the right leg and inability to walk without falling; onset had occurred two days before with fever, headache, pains in the back and legs, and general malaise.

Examination.—The temperature was 101, pulse 120, respiratory rate 20.

The skin, eyes, nose, throat, lungs, heart and abdomen were all entirely normal.

Examination of the right leg revealed the knee jerk reflex absent; the patient was unable to flex the thigh on the pelvis or flex the foot on the leg. Kernig's sign was positive. Oppenheim's and Babinski's signs were normal. There was no disturbance of sensation. Examination of the left leg revealed the knee jerk exaggerated. There was muscular tenderness throughout the thigh with normal flexion of the thigh on the pelvis and the foot on the leg. Kernig's sign was positive. Oppenheim's and Babinski's signs were normal or negative.

The patient was unable to stand on the right leg alone but could stand on both legs and walk with help with a slight dragging of the right foot and toe.

The arms, forearms and hands were normal. Function was good.

The posterior neck muscles were rigid and the patient was unable to flex the head on the chest without pain. There was no opisthotonos.

The pupils were of normal size and they reacted well to light and distance.

Ten cc. of clear spinal fluid was withdrawn; the pressure was slightly increased but returned to normal with this volume removed. Cell count in undiluted fluid was 100. Stained

smears showed 50 per cent polymorphonuclears and 50 per cent lymphocytes. No organisms were found with three Gram stains. Sugar was normal, a trace. Globulin was present with a heavy trace. Cultures were made on blood agar plates and reported at intervals of twelve, twenty-four, forty-eight and seventy-two hours.

The urine, March 23, was normal except for a slight trace of albumin. March 26 it was normal except for a slight trace of acetone. March 27 it was entirely normal.

March 23 there were 4,330,000 red blood cells, 85 per cent hemoglobin, 8,900 white blood cells, 64 per cent polymorphonuclears, 31 per cent lymphocytes, 4 per cent large mononuclears and 1 per cent basophils.

March 24 there were 4,210,000 red blood cells, 82 per cent hemoglobin, 9,750 white blood cells, 76 per cent polymorphonuclears, 29 per cent lymphocytes, 2 per cent large mononuclears and 1 per cent basophils.

March 25 there were 4,150,000 red blood cells, 82 per cent hemoglobin, 9,050 white blood cells, 70 per cent polymorphonuclears, 23 per cent lymphocytes, 5 per cent large mononuclears and 2 per cent basophils.

March 26 there were 4,150,000 red blood cells, 80 per cent hemoglobin, 6,450 white blood cells, 71 per cent polymorphonuclears, 22 per cent lymphocytes, 5 per cent large mononuclears and 2 per cent eosinophils.

March 27 there were 4,080,000 red blood cells, 80 per cent hemoglobin, 5,600 white blood cells, 70 per cent polymorphonuclears, 25 per cent lymphocytes, 4 per cent large mononuclears and 1 per cent eosinophils.

Treatment and Medication.—The patient entered the hospital March 22, 1939, at 9 p. m. The usual routine blood, urine and spinal puncture were done. Neoprontosil 15 grains (1 Gm.) every four hours for three doses was started at 11 p. m.

On the morning of March 23 examination was essentially the same as previously detailed, with the additional observation of toe drop with the foot extended and the thigh flexed on the pelvis on the right side, and paresthesias of both forearms and the little fingers of both hands. A molded plaster of paris splint was applied to the leg and foot at this time.

Sulfapyridine was started at 4 p. m., seven 7/10 grain tablets every two hours for twelve doses, at which time the temperature was 103.4 F., the pulse 130 and respiratory rate 28, with the blood picture 4,330,000 red blood cells, 85 per cent hemoglobin, 8,900 white blood cells, 64 per cent polymorphonuclears, 31 per cent lymphocytes, 4 per cent large mononuclears and 1 per cent basophils. The temperature returned to normal and remained so for twelve hours. The drug was temporarily discontinued, because the patient was slightly cyanotic and increasingly irritable, with anorexia, but was again started when the temperature returned to 100, in the same dosage, every three hours for six more doses. The temperature again returned to normal for a twelve hour period; the drug was discontinued, and the temperature returned to 99 for a two hour period only twice since sulfapyridine was stopped, and has been normal or subnormal for the past five days. The change in the blood picture with regard to giving sulfapyridine was as follows: March 23, 4,330,000 red blood cells, 8,900 white blood cells, 85 per cent hemoglobin. March 27, 4,080,000 red blood cells, 5,600 white blood cells, 80 per cent hemoglobin. The kidney function and urine were essentially normal at the onset and no untoward effects were noticed with the treatment. The slight rigidity of the neck muscles rapidly disappeared. The spinal fluid showed no growth on culture at twelve, twenty-four, forty-eight and seventy-two hours. The patient's general condition has satisfactorily improved. There is no further muscular paralysis than was present at the first visit, which was the third day after onset. The right leg and foot from the hip is in a molded splint, with no deformity, and the neurologic manifestations on this side are still an absent knee jerk and inability to flex the thigh on the pelvis or flex the foot on the leg.

CONCLUSIONS

This case could have terminated as it has in paralysis of the flexor muscles of the foot, leg and thigh of the right side without the use of sulfapyridine and not be unusual in any way; others have done it; but (1) the apparent dramatic response of the white blood cell count, (2) the drop in temperature from 104.5 to normal in twenty-four hours, remaining so for twelve hours and again responding to medication the second time, with continued approach to normal of the white blood cell count, and (3) the relief of the paresthesias, all so closely following the administration of sulfapyridine, leave us to determine whether this drug has a virtue in anterior poliomyelitis.

Take the report for what it is worth, but further use, properly given, is certainly justified in such cases, especially in patients seen early in the disease, as this case shows. I do not make the slightest claim of sulfapyridine being specific, having only a lingering hope for the future.

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HEMORRHAGIC MUCOCELE OF THE APPENDIX

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V. R., a high school girl aged 16, was admitted to the hospital Aug. 10, 1935. She had been in good health except for what she termed minor attacks of indigestion intermittently during the past two years. The attacks were never serious enough for her to consult her physician. On the morning of August 9 after arising she was taken with a dull aching pain in the right lower quadrant of the abdomen which did not radiate. The patient suffered profound nausea and vomited seven times during the day. There was no history of constipation and her menstrual periods were regular.

On admission to the hospital the patient's temperature was 100 F., the pulse 110 and the leukocyte count, 30,700. There was a definite palpable tumor mass in the right lower quadrant of the abdomen, exquisite tenderness over McBurney's point and an early peritoneal reflex. There was no abdominal distention; the last menstrual period had been normal and within ten days. A diagnosis of ovarian tumor with twisted pedicle was made and immediate operation advised.

Mucocele after removal, showing its tremendous size as compared with the appendix and its position lateral to the appendix.

Under ether anesthesia the abdomen was opened through a right paramesial incision and a tumor was exposed in the right iliac fossa. The tumor was entirely free of adhesions. The gallbladder and stomach and both kidneys were normal and there was no obstruction of the bowel. This tumor was dark in color and shaped like a cucumber. The appendix appeared to lie at its distal end and was attached approximately an inch from the junction of the appendix to the caput coli. The apparent pedicle was twisted about 120 degrees, and this whole mass was gangrenous. The tumor was removed from the appendix and the appendix was removed in the routine manner without drainage.

Convalescence was without incident and the patient was discharged in good condition on the eleventh postoperative day.

The gross appearance demonstrated a large, soft tumor with a thin, connective tissue wall almost bisected, thus forming two lobes filled with coagulated blood. The tumor was attached to the appendix. Section was made of the thin wall. The appendix, 6.5 cm. by 1 cm. at the proximal end, tapered to 6 mm. at the tip; the tumor was attached to the proximal portion by a small pedicle.

The microscopic section of the cyst wall filled with blood and showed hemorrhagic mucosa supporting only a few mucous glands, which showed hemorrhage into their lumens; the solitary follicles were enlarged, the lymphoid cells being pushed apart by edema and hemorrhage. The submucosa was edematous, its blood vessels were enlarged and congested and there was a large amount of hemorrhage into this coat; the muscular

Dr. Warren Howard permitted me to publish a report of this case. Dr. James B. Rucker gave his cooperation in the interpretation of the pathologic picture and Dr. George W. Bascom reviewed the literature.

coats were normal in thickness but were infiltrated with many lymphoid cells and a slight amount of hemorrhage.

The diagnosis was hemorrhagic mucocele of the appendix.

My interest in mucocele of the appendix was stimulated not only because of the rarity of this abnormality but also because it is a challenge to the diagnostic acumen of the general surgeon.

The frequency of the disease is of interest because, in reviewing the literature, I find that there have been approximately 250 cases reported since Virchow's[1] classic description of mucocele in 1863. Castle,[2] in a total of 13,158 autopsies, found twenty-eight cases, or a frequency of about 0.2 per cent. Mayo and Fauster[3] reviewed the surgical cases of the Mayo Clinic from 1917 to 1930 and found that mucocele of the appendix was encountered only seventy-six times in 31,200 cases in which appendectomy had been performed, a frequency of about 0.24 per cent.

A description of the pathologic changes occurring in the development of mucocele of the appendix is found in the work of Deaver,[4] and Vorhaus.[5]

The first process, according to Vorhaus, is assumed to be due to recurrent infections of the appendix causing a gradual stenosis of the lumen at one or more points distal to the obliterated lumen; sterile contents are presupposed. Then in the presence of actively secreting mucous glands a distention occurs, and theoretically there is a change in the mucus resulting in the transformation of mucin into pseudomucin.

The facts favoring this theory are as follows: There is a complete or almost complete stenosis of the lumen, or else with the increasing distention the fluid would be forced out. Secondly, there cannot be any pathogenic bacteria or else empyema or gangrene would result. Lastly, an active mucous membrane secreting mucus in excess of its absorption is necessary or distention would not occur. This results in the development of either rupture of the mucocele causing pseudomyxoma peritonaei or a thin walled watery cyst due to cessation of activity of the mucous glands which eventually becomes atrophic as a result of pressure atrophy. In my case the cystic fluid was hemorrhagic, probably owing to a ruptured blood vessel after the proximal portion of the appendix had become twisted.

Thus the rupture of a mucocele into the peritoneal cavity produces a condition of potential malignancy, pseudomyxoma peritonaei, the resulting effect on the patient and future response to treatment depending entirely on whether or not the mucous membrane lining the mucocele has been sufficiently destroyed by pressure atrophy.

The most common cause of pseudomyxoma peritonaei is due to pseudomucinous cystadenoma of the ovary. Therefore it is more frequent in the female, while mucocele of the appendix is the only known cause of pseudomyxoma peritonaei in the male.

The symptoms are vague and not characteristic of any specific clinical entity, especially the uncomplicated cases. Other than a history of (1) abdominal distress, (2) a palpable mass in the right lower quadrant of the abdomen, there are no other constant features. These points were the only constant factors mentioned by both Mayo and Fauster[3] and Dannreuther[6] in their reports. Although my patient had the symptoms of an acute surgical condition within the abdomen, which was due to a 120 degree rotation of the mucocele on the proximal portion of the appendix, these acute symptoms may be those of obstruction of the intestine, which has been reported to occur when the mucocele becomes invaginated into the cecum.

However, as a rule most of these patients tolerate these vague symptoms until they become so distressing and the pain so intensified that they voluntarily seek relief.

The diagnosis of mucocele of the appendix or pseudomyxoma peritonaei of appendical origin is usually made at autopsy, or at the operating table in a case with a preoperative diagnosis of ovarian tumor with or without a twisted pedicle, uterine fibroids, carcinoma of the cecum, terminal ileitis, intestinal obstruction or intraperitoneal neoplasm. In my case a diagnosis of ovarian tumor with twisted pedicle was made because of the acute onset of symptoms, the exquisite tenderness, the fever and the high leukocyte count. The disease is most common in middle aged and elderly women although my patient was only 16. There are no constant symptoms or signs which are pathognomonic of this disease. While roentgenographic evidence of mucocele of the appendix following a barium enema was used by Vorhaus,[5] Simon,[7] LeWald[8] and Lifvendahl and Ries[9] in the only preoperative diagnosis of mucocele of the appendix, this procedure does not always give constant results, since usually the lumen is stenosed or at best there may be an occasional mucocele with intermittent patency of the proximal end of the appendix.

However, with a history of vague gastrointestinal complaints and a palpable tumor mass in the right lower quadrant of the abdomen one should at least include mucocele of the appendix in the differential diagnostic considerations.

The treatment of mucocele of the appendix and pseudomyxoma peritonaei is definitely surgical. In the uncomplicated cases surgical removal is done with extreme caution to prevent rupture of the mucocele, while in complicated cases of mucocele of the appendix with pseudomyxoma peritonaei removal of the remaining parts of the ruptured mucocele and removal of as much of the mucoid gelatinous material and implants as possible are followed with roentgen therapy.

CONCLUSIONS

1. Mucocele of the vermiform appendix is a clinical rarity but is important because of its disastrous complications.
2. Its clinical incidence is approximately 0.24 per cent.
3. It is one of two known causes of pseudomyxoma peritonaei.
4. The clinical diagnosis is extremely difficult unless this condition is kept in mind, since there are no constant pathognomonic signs or symptoms especially incident to mucocele of the appendix.
5. The treatment is surgical.

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SULFANILAMIDE IN THE TREATMENT OF SMALLPOX

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I have recently treated seven cases of smallpox, four with sulfanilamide and three with symptomatic treatment:

CASE 1.—Mrs. G., aged 44, a housewife, white, first observed Jan 18, 1939, complained chiefly of severe backache and fever coming on rather suddenly during the preceding night. At first she felt chilly but did not have a definite rigor. This was accompanied with severe aching of the back in the lower lumbar region, across the hips and down the back of the legs. There was considerable headache and some nausea but no vomiting. There was some discomfort in the throat but no cough. There was no constipation, diarrhea or disturbance of the urinary system. There had been very little sweating. The patient was rather obese and appeared to be extremely ill. The skin was dry, the face was flushed and there was some restlessness. The temperature was 103.2 F.; the pulse rate 96 and the blood pressure 140 systolic, 90 diastolic. The eyes and nose were not congested but the throat showed red, cryptic, slightly enlarged tonsils and some redness of the pharynx. The chest was clear and there was no cardiac irregularity. There was no abdominal tenderness and no enlargement of the abdominal organs. The reflexes were normal. There was no rash on the skin. The urine showed a slight trace of albumin, with a high specific gravity but no pus cells, casts or erythrocytes.

The patient was given acetylsalicylic acid 5 grains (0.32 Gm.) every three hours; there was no question of her not staying in bed. The following day there was little change in the patient's condition other than a slight lowering of the temperature (101

F.). The acetylsalicylic acid seemed to ease the aching somewhat. On the third day the temperature was normal and the patient was feeling much better and was up about her household duties. On the fifth day the patient came to my office complaining of a "breaking out." She was covered—scalp, face, chest, arms, legs, palms and soles—with about 200 pustules about the size of a small pea, sitting on a red areolar base. On the palms and soles the pustules were very hard and tense. They contained a semipurulent material, which on smears showed short chained streptococci. On the seventh day definite umbilication was seen; on the ninth the pustules were beginning to dry, and on the thirteenth they were desquamating. The eruption was accompanied by a slight elevation of temperature (from 99 to 100.6 F.) and mild prostration.

CASE 2.—H. G., aged 18, the son of patient 1, a farmer, first seen February 5, complained chiefly of severe backache and headache, some nausea but no vomiting. The onset had been rather sudden the night before when a rather severe chill was experienced while he was attending a movie. There was no cough, constipation or symptom referable to the urinary tract. The symptoms followed the identical pattern of those presented by his mother. The patient was well developed and well nourished. The skin was dry and clear. The temperature was 104 F., the pulse rate 110 and the blood pressure 118/80. Otherwise the examination was negative. He was given acetylsalicylic acid 5 grains every three or four hours and sulfanilamide 10 grains (0.65 Gm.) every three hours. The following day the patient's condition was unchanged except for very slight cyanosis of the lips and some duskiness of the skin. There was no rash. On the third day the temperature was 99 F. and the patient felt much better. The aching had abated but there was some nausea. The dosage of sulfanilamide was reduced to 5 grains every four hours. On the fourth day the temperature was 98.2 F. and the patient was much improved. There were three very small dark red macules on the right forearm and three or four in the scalp. These were rather tender and caused some itching. On the sixth day the patient was out of bed and the following day he was attending to his farm work. The macules disappeared on the sixth day and there was no further eruption.

CASE 3.—Mr. F. G., aged 53, the husband of patient 1, a farmer, first seen February 8, complained chiefly of severe headache across the forehead; there was also some backache and generalized aching in the limbs. There were no other complaints. The temperature was 102 F., the pulse rate 96 and the blood pressure 138/96. The examination was otherwise negative. He was given acetylsalicylic acid 5 grains with phenobarbital one-half grain (0.032 Gm.), but this gave no relief and dilaudid 1/32 grain (0.002 Gm.) was given at intervals of about ten hours to relieve the aching. Marked restlessness and semidelirium also developed. On the third day the temperature, which had varied between 101 and 103 F., fell to 99 F., and the next day there was a widespread eruption of discrete red macules and papules about the size of a pea, the temperature became elevated again to 101 F., and the papules and macules in two days were well marked pustules, which soon became umbilicated. On the eleventh day of the disease they began drying up and on the fifteenth day they were practically all gone. The patient had been vaccinated forty years before. None of the other patients in this series had been vaccinated.

CASE 4.—Mrs. S., aged 30, daughter and neighbor of patient 1, a housewife, had been in and out of her mother's house while her mother was ill and became ill February 6 with symptoms very similar to those described in the foregoing cases, namely backache, headache, fever and some nausea. I first saw her February 7. The temperature ranged from 102 to 104 F. for three days and on the fourth day returned to normal. During this period she received sulfanilamide 10 grains every three hours and acetylsalicylic acid 5 grains as needed. On the fourth day a few small macules were noted on the wrists and forehead. The medication was discontinued on the fifth day, the macules disappeared on the sixth and she was about her household duties on the seventh.

CASE 5.—Mr. C., aged 34, a son-in-law of patient 1, a farmer, when first seen, February 8, had been ill for two days, complaining of intense backache, headache and high fever. The patient was well developed and well nourished and was apparently very ill. The temperature was 104 F. Acetylsalicylic acid 5 grains with phenobarbital one-half grain gave some symptomatic relief. On the fourth day the temperature was normal and no eruption was noted. On the fifth day, however, many macules were noted over the body which rapidly became papular, then vesicular and pustular, followed by umbilication on the ninth day of the disease and drying up and crusting on the fifteenth. The eruption, as in the other cases described, covered the entire body including the palms and soles, and there were some papules in the pharynx and nose.

CASE 6.—Mrs. C., aged 30, a daughter of patient 1, a housewife, was first seen February 18 after being ill about thirty-six hours. She had had a rather sudden onset, had complained of intense backache and headache and had vomited twice. The history and symptoms were identical with those in the preceding cases. Her temperature was 103 F. She was given sulfanilamide 5 grains every three hours and acetylsalicylic acid as needed for the aching. On the fourth day the fever subsided and on the sixth there were three well defined vesicles on the right cheek which went through the typical evolution of pustulation, umbilication and desquamation. During the last phase of the disease there was some slight elevation of temperature (from 99 to 100 F.). On the tenth day of illness she was out of bed and feeling fairly well.

CASE 7.—Mr. W., aged 30, white, a farmer, first seen February 23, had spent the night with patient 3 on February 15. During the night of February 22 he rapidly became very ill; he had a light chill that lasted fifteen minutes and aching of the back and limbs, and he vomited. When he was seen he was very restless. The temperature was 104.2 F. The pulse was 110. There were no significant changes found on examination other than the fever and the restlessness. Morphine one-fourth grain (0.016 Gm.) was required to relieve the backache. He was given sulfanilamide 10 grains every three hours. On the fourth day the temperature was normal and he was feeling very well. A few macules developed on the forearms which lasted about twenty-four hours. He was back at work on the seventh day.

COMMENT

These seven cases are all identical as to onset and the initial period, typical of smallpox. All show a sudden onset of severe and excruciating backache and headache with an abrupt rise in temperature persisting for three days. There seemed to be very little variation in the patients who received sulfanilamide and those who did not. However, in the patients who received sulfanilamide there was no pustular eruption except in case 6, in which three pustules occurred. In the cases in which sulfanilamide was not used there was the typical eruption of smallpox. Sulfanilamide seemed to abort the second phase of smallpox with its pustulation and scarring; there were some macular spots on all those receiving sulfanilamide, but they were evanescent. As far as morbidity goes, the patients who received sulfanilamide recovered immediately after the subsidence of fever and the symptoms of the initial phase. The effect of sulfanilamide in patients with the typical eruption already present is not known, since the patients who received sulfanilamide were started on the treatment before an eruption appeared.

The occurrence of these seven cases in the same community and mostly in the same family during an outbreak of smallpox, and the absence of an eruption in those who received sulfanilamide therapy does not seem to be coincidental.

SUMMARY

1. Three of the seven patients with smallpox were treated symptomatically and in all three the typical eruption of smallpox developed.

2. The remaining four patients were given sulfanilamide, and in three there was only an evanescent macular eruption which rapidly disappeared; in the remaining case there were only three pustules.

3. The patients who were given sulfanilamide were back at work about a week sooner than those who did not receive it.

4. Sulfanilamide seems to have a beneficial effect in the treatment of smallpox when administered early in the disease.

INTERSEXUALITY

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DETROIT

Intersexuality is an interesting phenomenon and cases of this type are not frequently encountered. In his book on genital abnormalities Young [1] has given a complete review of the subject and has presented cases of his own.

REPORT OF CASE

The following case is of interest:

E. Z., a student aged 12 years, was seen Sept. 26, 1932, by the late Dr. Max Ballin, having been referred by the Childrens' Center of Detroit because of abnormal sexual development.

At the time of the birth of the patient, the mother was told that there was some abnormality of the sex organs but that the baby's sex was female. The child was christened Ida and was reared as a girl. At the age of 10 a school nurse discovered that there was some abnormality of the sex organs but did not report the condition until the child was ready to enter high school at the age of 12. There had been no menstruation, either normal or vicarious. There was nothing significant in the past history.

The father is Polish and the mother is Lithuanian; both are living and are of average build.

The patient was well built and well nourished, of stocky type, with a height of 61½ inches (156 cm.), weighing 116 pounds (52.6 Kg.), with dark complexion and dark brown hair (fig. 1). The patient had a deep voice like a young man of 16 or 17. There was some hair on the upper lip. The breasts were of the flat male type. There was a female type of distribution of pubic hair. The penis was somewhat smaller than normal, hook shaped with chordee. Hypospadias of the scrotum, urethra and prepuce was present. The scrotal halves were fairly well developed and split in the middle and did not contain testicles (fig. 2).

Fig. 1.—Appearance of patient June 15, 1938. Note the short, stocky build with female distribution of pubic hair.

There was a rudimentary vagina 6 cm. in length and about 1 cm. below the penis (fig. 3). A well developed prostate could be felt by rectal examination. Physical examination was otherwise negative.

Laboratory examination revealed that the blood, urine and chemistry of the blood were normal; the Wassermann reaction was negative. The blood pressure was 110 systolic, 72 diastolic. The basal metabolism was plus 10 per cent.

X-ray examination of the skull was essentially negative; that of the chest and urinary tract was also negative. There was no suggestion of an adrenal tumor.

It was decided that the patient was of the male sex and he was therefore rechristened Eugene, he was sent away for two weeks, his hair was cut so that it conformed to the masculine type and he donned long trousers.

Beginning Dec. 22, 1932, Dr. Ballin performed a series of corrective plastic operations. In the first stage the chordee was corrected; a constricting band between the corpora cavernosa was excised which permitted straightening of the penis. Jan. 17, 1933, a plastic operation on the glandular part of the urethra was performed, and June 24 the fistula which had formed posterior to the glans was operated on. June 5, 1934, an anterior urethra was constructed from the skin flaps of the posterior aspect of the penis. This urethra was constructed over a rubber catheter but was not connected to the original

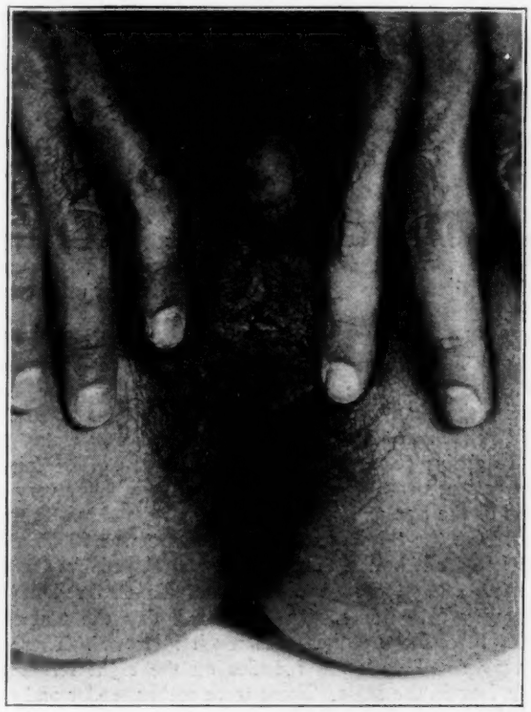

Fig. 2.—Showing scrotal halves with vaginal opening.

orifice of the bladder. Sounds were passed at periodic intervals every two or three weeks up to the present. His condition has been very satisfactory with this series of operations except for the fact that cystitis has persisted.

April 27, 1938, an acute appendicitis developed and on April 28 an acutely inflamed appendix was removed by one of us (Dr. Allen). The remainder of the abdomen was carefully examined and a small uterus was found with bilateral tubes and normally developed ovaries with the fimbriated ends of the tubes found patent. No rudimentary testicles were found. He made an uneventful recovery.

Gonadotropic assay of the urine was attempted several times but, because of bacterial infection present in the urine, all animals used in the tests died.

As he had reached the age of 18, a discussion with the patient concerning his psychosexual aspect was undertaken. He stated that he has always liked girls and for the last two years has had erections. He has had frequent orgasms with emissions, the discharge coming through the perineal urethra. Several specimens of this discharge were collected but no spermatozoa were found, nor were there any spermatozoa in the urine collected immediately after the discharge. He masturbated quite frequently and became sexually excited very easily. He expressed a desire to marry in the future but felt embarrassed because he had to sit down to urinate. This was his only complaint. He shaved once a week, for there was but a sparse growth of hair present on his face, being more like the "down of youth."

He was rather quiet in his speech, the voice being deeply masculine. He was of a retiring nature and it was difficult to have him discuss his case freely; he cooperated well in the examinations.

COMMENT

This patient would fall in the group termed by Klebs [2] as pseudohermaphroditismus masculinus internus. In this group, in addition to male sex organs, tubes, uterus and vagina have

From the Department of Medicine, Harper Hospital and Wayne University, and the Department of Surgery, Harper Hospital.

1. Young, Hugh: Genital Abnormalities, Hermaphroditism and Related Adrenal Diseases, Baltimore, Williams & Wilkins Company, 1937.

2. Klebs, cited by Young.[1]

also developed. According to Creevy,[3] one can classify any pseudohermaphrodite by the use of two adjectives: The first (masculine or feminine) indicates the nature of the sex gland. The second (external or internal, complete) indicates whether it is the sex of the internal or of the external or of both groups of genitalia that differs from that of the gonad.

Rubovits and Saphir,[4] in reporting their case of intersexuality, say: "Our microscopic evidence of testicular tubular atrophy and absence of spermatogenesis is enough to rule out the diagnosis of true hermaphroditism and to confirm again the fact that true hermaphroditism never has been found in man." Young,[1] on the other hand, states that his is the ninth accepted case of hermaphroditismus verus on record.

Psychically the patient considered himself as belonging to the male sex and was fond of the females. He says that he "feels mixed up" because he was reared as a girl and was "changed over." He has desires for intercourse with females, has erections and a discharge no doubt prostatic in origin, since no spermatozoa were ever found in the discharge or in the urine passed immediately after the discharge. No trace of a testicle could be found at operation.

His build is distinctly masculine, as shown in figure 1. His voice is deeply masculine and was present at the age of 12, indicating a well developed cartilaginous larynx. He is only 63 inches (160 cm.) in height, showing that the union and

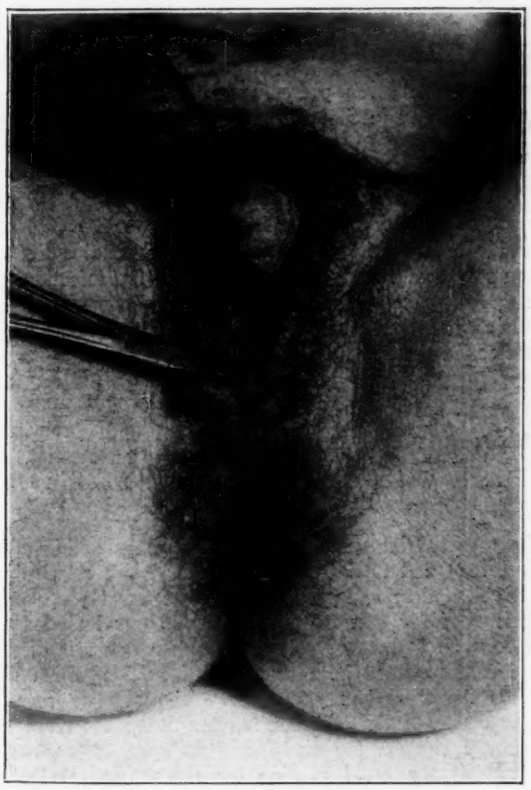

Fig. 3.—Forceps in vaginal opening.

ossification of the epiphyses took place prematurely. His appearance is very much like that seen in achondroplastic dwarfs with short muscular arms and legs.

SUMMARY

The case of intersexuality here reported is classified according to the Klebs grouping as pseudohermaphroditismus masculinus internus. In this group the male individual has also tubes, a uterus and a vagina. Our patient had, in addition, well developed ovaries. No testicles were seen or palpated.

The prostate was well developed. The patient had been reared as a girl until the age of 12 years, when he was rechristened with a masculine name and has since lived as a male. His sexual inclination is toward the female sex. He has erections and orgasms with a prostatic discharge through a perineal urethra. Micturition can be accomplished only in the sitting position.

964 Fisher Building.

3. Creevy, C. D.: Pseudohermaphroditism: Report of Five Cases, Internat. S. Digest **16**: 195-212 (Oct.) 1933.
4. Rubovits, W. H., and Saphir, William: Intersexuality, J. A. M. A. **110**: 1823-1826 (May 28) 1938.

DEATH FROM GRANULOCYTOPENIA AFTER SULFANILAMIDE THERAPY

PHILIP CORR, M.D., AND RALPH N. ROOT, M.D., RIVERSIDE, CALIF.

Sulfanilamide is such a valuable drug that it is being used very widely and with perhaps some lack of caution. For this reason it seems wise to report another fatal case to the small series of severe granulocytopenias resulting presumably from its use.

REPORT OF CASE

Mrs. M. S., white, American, aged 22, entered the Riverside County Hospital Sept. 19, 1938, because of a severe throat infection of about two weeks' duration. There had been a high fever with vomiting for two days. At the time of entrance into the hospital there was no other relevant history.

On physical examination at that time the patient was well developed and well nourished and appeared acutely ill. The temperature was 104.6 F., the pulse 136 and the respiratory rate 28. A general physical examination was done, examination of the vagina and rectum being omitted; the following observations were made: There were several superficial ulcerated areas about the lips. A few white patches were noted on the gums. The breath was foul. The tonsils were moderately enlarged and covered with a grayish, necrotic membrane. No bleeding was noted on traumatization of the membrane. The pharynx was injected. There was a mild anterior cervical adenitis bilaterally. On the posterior surface of the left hand between the first and second fingers was a small linear abrasion, surrounded by a small area of inflammation.

A smear from the throat revealed many diphtheria-like organisms. On the basis of this report 65,000 units of diphtheria antitoxin was given promptly. The following day culture of material from the throat was negative for diphtheria and the smear was reported negative for diphtheria. Numerous fusiform bacilli with a few spirilla were noted.

On the day of entrance the laboratory reported the hemoglobin content to be 68 per cent, with 3,120,000 red blood cells, 800 white blood cells with 8 per cent polymorphonuclear neutrophils, 2 per cent eosinophils and 90 per cent lymphocytes. The condition was then recognized as agranulocytic angina. Pentnucleotide was given intramuscularly in four doses of 10 cc. between meals on September 20 and 21. A transfusion of 550 cc. of whole citrated blood was given September 21.

September 20, the day after admission to the hospital, the white blood cell count improved to 1,100 per cubic centimeter with 48 per cent lymphocytes and 52 per cent polymorphonuclear neutrophils. The following day, the day of the patient's death, the white blood cell count was 2,000 with 56 per cent lymphocytes and 44 per cent neutrophils.

The patient's temperature remained around 103.6 to 105 F. until two days after her entrance into the hospital, when it gradually rose to 107.8 (rectal) before death. She was delirious much of the time the last day. At the time of death the small inflammatory lesion of the left hand amounted to a cellulitis involving most of the left hand posteriorly.

After repeated questioning by both of us the patient finally admitted, on the day of her death, that she had taken some tablets for leukorrhea. After this admission it was found that on Aug. 27, 1938, she had taken 80 grains (5 Gm.) of sulfanilamide, repeating this dose the following day. She then took 40 grains (2.6 Gm.) a day for six days and then 20 grains (1.3 Gm.) a day for seven days, the last tablets being taken September 11, eight days before she entered the hospital. Altogether 540 grains (35 Gm.) was given in a period of fifteen days by an osteopathic physician. No check was made of the blood count until entrance into the hospital, and no mention

was made by this practitioner of the medicine given when he sent the patient to the county hospital as probably having diphtheria.

The Kahn test was strongly positive, the report being made the day after the patient's death. A blood culture was negative after ten days' incubation. The urinalysis on entrance revealed a heavy trace of albumin, with a negative sugar reaction, from 6 to 8 white blood cells per high power field, and a markedly positive acetone reaction.

Autopsy was done by one of us (R. R.) on the embalmed body sixteen hours after death. There was a soft, nonpitting swelling of the dorsum of the left hand. There was an abrasion between the first and second fingers of the left hand with considerable dark bluish discoloration in that region. There was moderate swelling of the neck with palpable lymph nodes bilaterally. No other lymphadenopathy was noted.

When the pleural cavity was opened no free fluid was encountered, and no adhesions were noted. The pleurae showed evidence of some inflammation. There were several dark purplish hemorrhagic areas seen on the surface of the lungs, most marked in the right upper lobe. These were from 1 to 5 cm. in diameter and on cut section appeared dry, rough and raised. They were not limited to the surface and were not wedge shaped. There was some evidence of necrosis present in the centers. Otherwise the lungs were essentially normal. The pericardium was markedly injected and there were several small hemorrhagic areas. No abnormal amount of fluid was seen in the pericardial sac. The heart was not enlarged. The heart muscle was firm and of good tone, while its chambers, valves and vessels showed no abnormalities.

No free fluid was found in the abdominal cavity. The peritoneum was white and glistening. No adhesions were present. The liver appeared congested and there was a small dark area noted on the anterior surface of the right lobe which was soft and necrotic on cut section, but no free pus was present. The spleen was soft and moderately enlarged and had a smooth capsule. There was no evidence of abscess or of infarction. The stomach was dilated but essentially normal. The small and large intestine showed no abnormalities. The kidneys were moderately enlarged. Thin capsules stripped with ease, revealing congested surfaces. The parenchyma was poorly outlined. There was no dilatation of the ureters. No inflammation of the bladder was noted. The tubes and surrounding tissues were markedly congested and thickened, but no exudate was present. The ovaries were essentially normal. The uterus was small.

Sections of tissue from various organs were removed and examined by Dr. Oran I. Cutler of Loma Linda. His report showed the following:

Microscopic Examination.—The wall of the fallopian tube was congested but there was practically no scarring and no leukocytic infiltration was present. There was edema of the lung, and fairly numerous heart disease cells were present. In one section there were many masses of bacteria but no leukocytic reaction. In the ovaries there were some cysts lined with tiny dark cells. The kidney showed no definite pathologic changes. The pulp of the spleen was loose and relatively acellular. The liver showed some cloudy swelling.

The final diagnosis was agranulocytic angina caused presumably by sulfanilamide.

There was serologic evidence of syphilis.

COMMENT

Nine deaths have previously been recorded in the literature from agranulocytosis apparently caused by sulfanilamide according to a compilation by Kracke [1] in October 1938. A few more cases have been recorded in which recovery has taken place. Many more have probably occurred—some unrecognized, some unreported. With the increasing use of this valuable drug, more cases may be expected.

It is assumed that the drug, added to some unknown factor, produces a maturation arrest of the granulopoietic tissues.

There should be some simple means of avoiding this grave complication. One suggestion is commonly made [2] to have repeated frequent blood counts. This is an excellent suggestion and should be given serious consideration. However, if followed rigidly and as a routine by all those giving sulfanilamide it would probably cost many more lives by restriction of the use of sulfanilamide than it would save by prevention of agranulocytic deaths, which are apparently very rare. In the case of agranulocytosis from aminopyrine, it is relatively easy to solve the problem by substituting some other similar drug. As yet we have not found a comparatively effective, relatively harmless substitute for sulfanilamide.

Even with frequent blood counts it has not been proved that this will prevent deaths from agranulocytosis. In Young's [3] case examination of the blood five days before death failed to reveal an impending granulocytopenia, although the white blood count was reduced from 12,000 to 7,800. The granulocytopenia developed in this case four days after the sulfanilamide was discontinued.

A second suggestion has been made which should receive serious consideration, i. e. that any patient having toxic symptoms of any nature should (1) be denied further use of the drug or (2) should be watched very closely. This would seem to be an excellent rule to follow. Further use of the drug should be denied if the condition being treated is not of major seriousness, and the policy of strict observation should be enforced with blood counts every other day if sulfanilamide therapy is urgent.

A third point in preventing agranulocytic complications might be made concerning the dosage. In an adult the smallest dose of sulfanilamide given preceding the onset of agranulocytosis was 25 Gm., and in that case it was probable that the patient had taken more. In practically all the other cases reported 35 Gm. or more had been ingested in a period of two weeks or more. With our present information we might say that the general practitioner might safely give up to 20 Gm. of sulfanilamide before being concerned about agranulocytosis. That dosage may well care for many of the simpler infections treated with this drug.

This case further substantiates the causal relationship between sulfanilamide therapy and agranulocytosis.

1. Kracke, R. R.: Relation of Drug Therapy to Neutropenic States, J. A. M. A. **111**: 1255-1259 (Oct. 1) 1938.
2. Sulfanilamide and the Blood, editorial, Ann. Int. Med. **12**: 725 (Nov.) 1938.

NICOTINE POISONING WITH A SALT OF NICOTINE

A. F. WAGNER, M.D., AND MILES E. DRAKE, PH.D.
LOS ANGELES

The reported instances of fatal nicotine poisoning according to Webster [1] and to Beeman and Hunter [2] are relatively infrequent. The nicotine insecticides are the usual preparations of nicotine used for suicidal purposes. To our knowledge there have been no reports of poisoning with a dry salt of nicotine.

The case reported here was one of suicide which came to the Los Angeles County coroner. The autopsy revealed no evident cause of death. The viscera were therefore sent to the county chemist for analysis.

Following the usual routine for extracting alkaloids there was left, after evaporation of the alkaline ether extract of the stomach contents, a considerable quantity of white crystalline material. This white residue was purified and proved to be nicotine by the mercury bichloride and gold chloride microchemical tests and this was confirmed by biologic tests on frogs. Chemical examination showed that the nicotine salt was not the sulfate or hydrochloride but the tartrate. Later crystals of nicotine tartrate were identified in some cigarets found among the effects of the deceased. As the patient was a pharmaceutical chemist and had access to the salts of nicotine, one may readily understand how such a little used salt of nicotine was employed in this instance.

3. Young, C. J.: Agranulocytosis and Para-Amino-Benzene-Sulfanilamide, Brit. M. J. **2**: 105 (July 17) 1937.

Dr. Wagner is autopsy surgeon, Los Angeles County; Dr. Drake is assistant to the county chemist.

1. Webster, R. W.: Legal, Medicine and Toxicology, Philadelphia, W. B. Saunders Company, 1930, p. 618.
2. Beeman, J. A., and Hunter, W. C.: Fatal Nicotine Poisoning, Arch. Path. **24**: 481 (Oct.) 1937.

Special Article

TYPHOID IN THE LARGE CITIES OF THE UNITED STATES IN 1938

TWENTY-SEVENTH ANNUAL REPORT

The means employed for obtaining data for this report were described last year. A communication was addressed to the health officer of each of the ninety-three cities, requesting the number of deaths from typhoid both among residents and among nonresidents which were recorded for 1938. The difficulty of obtaining an adequate estimate of population continues and will harass the public health official until the 1940 census has been completed. In all instances the local estimate when furnished by the health officer has been used. There are some who feel that their cities have grown slightly during the past years and others take credit for a decrease in population. Study indicates that in no instance is a grave statistical error committed by using the local estimate rather than falling back on the 1930 census figures. It is hoped that after the 1940 figures become available these tables can be revised, but even then it is anticipated that the corrections will be of only a minor character.

TABLE 1.—*Death Rates of Fourteen Cities in New England States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Bridgeport	0.0	0.0	0.0	0.3	0.5	2.2	4.8	5.0	10.3
Fall River	0.0	0.0	0.9	0.2°	2.2	2.3	8.5	13.4	13.5
Lynn	0.0	0.0	1.0	0.2	1.5	1.6	3.9	7.2	14.1
New Bedford	0.0	0.0	1.8	1.1°	1.5	1.7	6.0	15.0	16.1
Providence	0.0	0.4	0.8	1.1	1.3	1.8	3.8	8.7	21.5
Cambridge	0.0	0.8	0.0	0.9	2.1	4.3	2.5	4.0	9.8
Lowell	0.0	1.0	1.0	1.0°	2.6	2.4	5.2	10.2	13.9
Worcester	0.5*	0.5*	0.0	0.6	1.0	2.3	3.5	5.0	11.8
Hartford	0.5*	1.1	0.5	1.2	1.3	2.5	6.0	15.0	19.0
Boston	0.6†	0.4*	0.1*	0.6	1.2	2.2	2.5	9.0	16.0
Springfield	0.7*	0.7*	0.0	1.0	0.4	2.0	4.4	17.6	19.9
Waterbury	0.9	0.0	0.0	0.4	1.2	1.0	8.0	18.8	....
Somerville	1.0	0.0	0.0	0.4	1.3	1.6	2.8	7.9	12.1
New Haven	1.2†	1.2	1.2	0.7	0.6	4.4	6.8	18.2	30.8

* All typhoid deaths were stated to be in nonresidents.
° Rate computed from population as of April 1, 1930, as no estimate for July 1, 1933, was made by the Census Bureau.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.

Paratyphoid has again been excluded. In tables 1 to 8 inclusive (as well as in table 10) a special note has been made of cities in which all deaths occur among nonresidents. Similar notation has been added for the death rates for 1936 and 1937 but in no instance for preceding years. Another symbol has been used to indicate those cities in which more than one third of the reported deaths were stated to have been among nonresidents.

Special attention is called to the cities listed in table 9 which report no typhoid death during the past two years. Bridgeport reports no death in five years, Fort Wayne no death in four years, South Bend and Utica no death in three years. Attention is also directed to the seven cities (Toledo, Hartford, Syracuse, Worcester, Springfield, Evansville and Camden) which are placed among those of first rank rather than on the honor roll in table 10 merely because they have been charged with deaths among nonresidents. It is quite appropriate to think of these cities as belonging in the honor group. Local circumstances beyond the control of the public health officer frequently require the hospitalization in large cities of cases which belong in the surrounding rural communities. This same comment applies to those cities in which a relatively high share of the typhoid deaths occurred among nonresidents.

Seven of the large New England cities report no death from typhoid in 1938 (table 1). Only four of these cities (Bridgeport, Fall River, Lynn, New Bedford) were in this same honor group in 1937. Bridgeport has extended its good record to five years. Somerville, which last year recorded no death for four years in succession, reports one death among residents in 1938. Springfield, after passing through a period of three successive years with no death among residents, reports one death among nonresidents in 1938. Providence reports no death in 1938 among twelve resident patients. Three of these cases occurred in one family and are attributed to the fact that children were playing near an old privy. Of two deaths reported from New Haven, one occurred in a nonresident. Of five deaths reported from Boston, three were among nonresidents. Hartford, with but one death and that in a nonresident, reports that in 1936, during the Connecticut River flood, 97,334 typhoid inoculations were given and that during the 1938 flood 4,435 such protective treatments were administered to Hartford residents. The New England cities as a whole (population 2,645,438), although reporting a rate as low as that for 1937, have lost first place among the grouped cities (table 13).

TABLE 2.—*Death Rates of Eighteen Cities in Middle Atlantic States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Utica	0.0	0.0	0.0	0.6	1.1	3.9#	...	....	....
Reading	0.0	0.0	1.8	0.4	1.6	6.0	10.0	31.9	42.0
Buffalo	0.0	0.2*	0.3	0.6	2.7	3.9	8.1	15.4	22.8
Scranton	0.0¶	0.7*¶	0.0¶‡	1.4	1.8	2.4	3.8	9.3	31.5
Erie	0.0	0.8*	0.8*	1.0	0.9	2.3	6.9	49.0	46.6
Elizabeth	0.0	0.8	0.8	0.9	1.6	2.4	3.3	8.0	16.6
Rochester	0.3	0.0	0.6†	0.4	1.7	2.1	2.9	9.6	12.8
New York	0.3	0.3	0.4	0.8	1.3	2.6	3.2	8.0	13.5
Newark	0.4	0.0	0.2	0.3	0.9	2.3	3.3	6.8	14.6
Syracuse	0.5*	0.0	0.0	0.8	0.8	2.3	7.7	12.3	15.6
Paterson	0.7	0.0	0.7	0.9	1.0	3.3	4.1	9.1	19.3
Yonkers	0.7	0.0	0.7‖	0.7	0.5	1.7	4.8	5.0	10.3
Philadelphia	0.7¶	1.4	0.7	0.9	1.1	2.2	4.9	11.2	41.7
Jersey City	0.9	0.3	0.6*	0.2	0.9	2.7	4.5	7.2	12.6
Pittsburgh	0.9†	0.7†	0.7†	0.9	2.4	3.9	7.7	15.9	65.0
Albany	1.4	1.3†	1.5	1.1	1.8	5.6	8.0	18.6	17.4
Camden	1.6*	1.6	0.8	2.8	4.4	5.9	4.9	4.5	4.0
Trenton	2.4†	0.8*	0.8*	1.1	2.1	8.2	8.6	22.3	28.1

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents
# Incomplete data.
¶ Typhoid deaths furnished by Pennsylvania Department of Health, Harrisburg.
‡ Rate computed from 1930 census population, as no local estimate was given.
‖ Corrected rate. In review for 1936, Yonkers reported cases instead of deaths.

The lowest rate, of 0.35, is reported from the East North Central states, while second place goes to the West North Central cities and third place to the Middle Atlantic cities. In the New England cities there were recorded twelve deaths in 1938, a number identical with that of 1937.

The Middle Atlantic states have a group rate (0.44) which is significantly lower than that of 1937 (0.51). The 1938 rate is slightly below that of the New England

The preceding articles in this series were published in THE JOURNAL May 31, 1913, p. 1702; May 9, 1914, p. 1473; April 17, 1915, p. 1322; April 22, 1916, p. 1305; March 17, 1917, p. 845; March 16, 1918, p. 777; April 5, 1919, p. 997; March 6, 1920, p. 672; March 26, 1921, p. 860; March 25, 1922, p. 890; March 10, 1923, p. 691; Feb. 2, 1924, p. 389; March 14, 1925, p. 813; March 27, 1926, p. 948; April 9, 1927, p. 1148; May 19, 1928, p. 1624; May 18, 1929, p. 1674; May 17, 1930, p. 1574; May 9, 1931, p. 1576; April 30, 1932, p. 1550; May 13, 1933, p. 1491; May 19, 1934, p. 1677; June 8, 1935, p. 2093; June 6, 1936, p. 1983; June 19, 1937, p. 2118, and July 30, 1938, p. 414.

cities and is nearly half the rate of the Middle Atlantic cities for the quinquennial period 1931-1935 (0.80). There has been no death recorded in Utica for the past three years. Syracuse continued to hold her place of honor in that there was recorded no death among

TABLE 3.—*Death Rates of Nine Cities in South Atlantic States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Norfolk	0.8	0.8	0.0	4.2	2.2	2.8	8.8	21.7	42.1
Atlanta	0.9†	1.9†	3.2†	7.2	11.1	14.5	14.2	31.4	58.4
Washington	1.0†	1.9	1.6	2.6	2.8	5.4	9.5	17.2	36.7
Baltimore	1.5†	1.2	0.9	1.4	3.2	4.0	11.8	23.7	35.1
Tampa	2.0†	0.0	0.0	3.0	3.8	19.1	43.9#	....	....
Wilmington	2.7	1.8	0.9	1.5	3.1	4.7	25.8#	23.2#	33.0
Richmond	2.7†	3.2†	2.7	2.5	1.9	5.7	9.7	15.7	34.0
Miami	3.6†	6.3	3.1†	2.2	3.5	...	...	....	....
Jacksonville	5.2	4.0	1.3	1.7	4.4	...	...	....	....

Incomplete data.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.

residents in 1938, but the record of this city has been somewhat embarrassed by the occurrence of one typhoid death among nonresidents in 1938. Six cities in this group report no typhoid death in 1938. There were seven such cities in 1937. Utica and Reading are the only two cities reporting no death in each of these two years. Buffalo, Scranton, Erie and Syracuse record no death among residents during the past two years. Scranton, as in the case of Springfield, has passed through five consecutive years with no death among residents. Paterson, with no death in 1937, records one death among residents in 1938. Newark, with no death in 1937, reports two among residents in 1938. New York reports twenty-two deaths, of which twenty were among residents. Pittsburgh reports six deaths, three among residents. Philadelphia reports fifteen deaths, all among residents. In the group as a whole (population

TABLE 4.—*Death Rates of Eighteen Cities in East North Central States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Fort Wayne	0.0	0.0	0.0	2.2	4.2	12.9	7.3	....	....
South Bend	0.0	0.0	0.0	0.7	...	...	...	....	....
Milwaukee	0.0	0.0	0.3	0.2	0.8	1.6	6.5	13.6	27.0
Canton	0.0	0.0	1.0°	0.9	1.4	3.3	8.9	....	....
Grand Rapids	0.0	1.0*	0.6*	0.2	1.0	1.9	9.1	25.5	29.7
Youngstown	0.0	1.1	1.1†	1.1	1.1	7.2	19.2	29.5	35.1
Peoria	0.0	1.7*	1.7*	0.9	0.2	3.7	5.7	16.4	15.7#
Detroit	0.2†	0.3	0.5	0.6	1.3	4.1	8.1	15.4	22.8
Chicago	0.3	0.3	0.3	0.4	0.6	1.4	2.4	8.2	15.8
Cleveland	0.3†	0.5	1.0	1.1	1.0	2.0	4.0	10.0	15.7
Toledo	0.3*	1.2*	1.0†	1.3	3.0	5.8	10.6	31.4	37.5
Columbus	0.3	1.5	3.7*	2.0	2.1	3.5	7.1	15.8	40.0
Akron	0.4	1.6†	0.8†	0.8	1.5	2.4	10.6	21.0	27.7#
Cincinnati	0.6	1.3†	1.9†	1.4	2.5	3.2	3.4	7.8	30.1
Evansville	0.9*	2.7†	0.0	1.9	6.2	5.0	17.5	32.0	35.0
Flint	1.2	3.0†	1.2	0.7	1.6	4.6	22.7	18.8	46.9
Indianapolis	1.3	1.3	0.8†	1.2	2.7	4.6	10.3	20.5	30.4
Dayton	1.3†	1.4†	1.8†	0.8	1.9	3.3	9.3	14.8	22.5

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.
° Rate computed from 1930 census population, as no local estimate was given.

13,469,996) there were fifty-nine deaths in 1938 compared with sixty-eight in the preceding year. There are two geographic areas which have a lower death rate for 1938, the East North Central and the West North Central.

The rate for the South Atlantic cities (1.74) is much improved over that of 1937 (1.96) but is not as low as that of 1936 (1.55). In these cities (population 2,638,626) there occurred forty-six deaths in 1938 and fifty-one in 1937. There was no city without a death in 1938, although six of the nine cities in this group report that one third or more of the deaths occurred among nonresidents. Baltimore reports thirteen deaths, eight among residents. It is reported that, in investigating sources of infection in typhoid cases, there were located sixteen carriers presumably responsible for twenty cases. Many cities in the South continue to be embarrassed by the high percentage of deaths among nonresidents. The large cities serve as hospital centers for the neighboring suburban and rural areas which are without adequate means of caring for the communicable diseases and which frequently suffer from the want of whole-time local public health supervision. Of five deaths recorded for Richmond, four were

TABLE 5.—*Death Rates of Six Cities in East South Central States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Louisville	0.8†	0.6*	1.4†	2.3	3.7	4.9	9.7	19.7	52.7
Birmingham	1.7†	1.4	5.0°	3.9	8.0	10.8	31.5	41.3	41.7
Chattanooga	1.7	1.7	0.0	4.7	8.0	18.6	27.2	35.8#	....
Memphis	3.1†	4.9†	4.7†	7.9	9.3	18.9	27.7	42.5	35.3
Knoxville	3.9	3.2	4.1	5.7	10.7	20.8	25.3#	....	....
Nashville	4.9†	1.2*	4.4†	5.6	18.2	17.8	20.7	40.2	61.2

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.
° Rate computed from 1930 census population, as no local estimate was given.

TABLE 6.—*Death Rates of Nine Cities in West North Central States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Wichita	0.0	0.0	1.9	0.7	1.2	6.3	...	....	....
Minneapolis	0.0	0.2*	0.0	0.8	0.8	1.9	5.0	10.6	32.1
Kansas City, Kan.	0.0	0.8*	2.3	1.1	1.7	5.0	9.4	31.1	74.5#
Omaha	0.0	1.3	0.9*	0.9	1.3	3.3	5.7	14.9	40.7
St. Louis	0.5	1.1†	0.8	1.6	2.1	3.9	6.5	12.1	14.7
St. Paul	0.7	0.0	0.7	0.7	1.4	3.4	3.1	9.2	12.8
Des Moines	0.7	0.7	2.8	2.1	2.4	2.2	6.4	15.9	23.7
Kansas City, Mo.	0.7†	1.4†	0.5	1.5	2.8	5.7	10.6	16.2	35.6
Duluth	1.0	0.0	0.0	1.0	1.1	1.7	4.4	19.8	45.5

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.

among nonresidents; of six in Washington, two were among nonresidents. Three deaths among nonresidents are included among the five deaths reported for Miami. It is stated that for one nonresident death the infection was contracted prior to the arrival of the patient in Florida. Miami originally reported eight deaths for the year 1937. The director of health states that this should now be changed to six deaths for that year. Of eight deaths recorded by Jacksonville, but one occurred in a nonresident. The health department reports that a plan is being developed to require inoculation of all food handlers against typhoid. This city continues to serve as a medical center for seven or eight nearby rural counties with no public health units. Such a unit has, however, recently been established in Duval County, which includes the territory immediately surrounding Jacksonville. After two consecutive years with no typhoid death, Tampa records one death among residents for two reported in 1938.

The East North Central cities (population 9,907,440) can boast of the best record for 1938 (rate 0.35). In 1937 there were sixty-one deaths reported from these

cities. In 1938 the number of deaths had dropped to thirty-five. There are seven cities which report no death and two additional cities (Toledo, Evansville) which record no death among residents. In addition to this there are three cities (Detroit, Cleveland, Dayton) in which one third or more of the reported cases were stated to be in nonresidents. Fort Wayne reports no typhoid death in four years, South Bend no death in three years, Milwaukee and Canton no death in two years. There has been no death among residents in Grand Rapids and Peoria during the past four years. Toledo reports no resident death in two years. Dayton records a significantly high proportion of deaths among nonresidents during each of the past three years. Of three deaths each in Cleveland and Detroit, one in each

TABLE 7.—*Death Rates of Eight Cities in West South Central States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Tulsa	0.6	0.0	0.7‡	1.1	8.3	16.2#	...	....	....
Oklahoma City	1.3	3.1	4.3‡	4.3	7.4#	...	...	....	....
San Antonio	2.3†	3.8	2.7	4.2	4.6	9.3	23.3	29.5	35.9
Fort Worth	2.7†	2.2	3.3	4.6	5.9	6.1	16.3#	11.9	27.8
Houston	3.1	2.0	3.8	3.2	4.8	7.6	14.2	38.1	49.5#
Dallas	4.3†	3.0*	1.5*‡	5.4	7.3	11.2	17.2	....	....
New Orleans	5.5†	2.3†	6.5†	9.6	9.9	11.6	17.5	20.9	35.6
El Paso	5.9	0.0	6.8‡	4.9	9.1	10.8	30.7	42.8	....

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.
‡ Rate computed from 1930 census population, as no local estimate was given.

TABLE 8.—*Death Rates of Eleven Cities in Mountain and Pacific States from Typhoid per Hundred Thousand of Population*

	1938	1937	1936	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Seattle	0.0	0.0	0.5	0.6	2.2	2.6	2.9	5.7	25.2
Tacoma	0.0	0.0	0.9	0.7	1.8	3.7	2.9	10.4	19.0
Portland	0.0	0.3	0.6	0.8	2.3	3.5	4.5	10.8	23.2
Salt Lake City	0.0	0.7	0.0	1.0	1.9	6.0	9.3	13.2	41.1
San Diego	0.0	1.6†	1.8†	1.3	1.0	1.6	7.9	17.0	10.8
Long Beach	0.6	0.0	0.6*	0.2	1.1	2.1#	...	....	....
Oakland	0.6†	0.3	0.3	1.0	1.2	2.0	3.8	8.7	21.5
Los Angeles	0.6†	0.7†	1.0†	0.8	1.5	3.0	3.6	10.7	19.0
San Francisco	0.7	0.6	0.3†	0.8	2.0	2.8	4.6	13.6	26.3
Denver	1.0	2.7	2.0	1.8	2.6	5.1	5.8	12.0	37.5
Spokane	2.4†	0.0	0.8	1.4	2.2	4.4	4.9	17.1	50.3

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.

city was of a nonresident. Chicago reports a total of twelve deaths, two of which were stated to be in nonresidents.

The six cities in the East South Central Group show an increase in the death rate (2.10 in 1937, 2.36 in 1938). This rate, however, is considerably below that of 1936 (3.35) and that of the quinquennial period 1931-1935 (4.81). The actual number of deaths in these six cities increased from twenty-eight in 1937 to thirty-two in 1938. No city in this group is without a death in 1938. The influence of the surrounding rural areas in contributing to the hospital load and thus to the death rate of the urban centers in the South is well illustrated by the fact that in Birmingham, Memphis and Nashville more than one third of the deaths were among nonresidents. Birmingham, with five deaths, reports two in nonresidents; Memphis, with nine deaths, records four in nonresidents; Nashville, with eight deaths, reports three in nonresidents. Chattanooga reports two deaths among residents, with a comment that in both instances the source of infection was outside the city. Special mention of the influence of the surrounding rural area and the Negro population to the death rate for Memphis was made in our report a year ago.

TABLE 9.—*Thirteen Cities with No Typhoid Death in 1937 and 1938*

Bridgeport*	Lynn	South Bend†
Canton	Milwaukee	Tacoma
Fall River	New Bedford	Utica†
Fort Wayne**	Reading	Wichita
	Seattle	

* No typhoid death in five years.
** No typhoid death in four years.
† No typhoid death in three years.

The West North Central group (population 2,785,688) reports a noteworthy reduction in the death rate (0.76 in 1937, 0.40 in 1938). There is but one group of cities (East North Central) which has a lower death rate. Four cities (Wichita, Minneapolis, Kansas City, Kan., Omaha) report no typhoid death in 1938 and the first three of these record no death among residents in 1937. Minneapolis has had no death in a resident during the past three years. Duluth, after two years with no death, now reports a single death in a resident. This death, however, is sufficient to move Duluth from the head to the bottom of the table. This is the only group in which there is no city with a death rate in

TABLE 10.—*Death Rates from Typhoid in 1938*

Honor Roll: No Typhoid Deaths (Twenty-Nine Cities)

Bridgeport	Lowell	Reading
Buffalo	Lynn	Salt Lake City
Cambridge	Milwaukee	San Diego
Canton	Minneapolis	Scranton
Elizabeth	New Bedford	Seattle
Erie	Omaha	South Bend
Fall River	Peoria	Tacoma
Fort Wayne	Portland	Utica
Grand Rapids	Providence	Wichita
Kansas City, Kan.		Youngstown

First Rank: from 0.1 to 1.9 Deaths per Hundred Thousand (Forty-Eight Cities)

Detroit	0.2†	Los Angeles	0.6†	Pittsburgh	0.9†
Chicago	0.3	Oakland	0.6†	Waterbury	0.9
Cleveland	0.3†	Tulsa	0.6	Denver	1.0
Columbus	0.3	Des Moines	0.7	Duluth	1.0
New York	0.3	Kansas City, Mo.	0.7†	Somerville	1.0
Rochester	0.3	Paterson	0.7	Washington	1.0†
Toledo	0.3*	Philadelphia	0.7	Flint	1.2
Akron	0.4	San Francisco	0.7	New Haven	1.2†
Newark	0.4	Springfield	0.7*	Dayton	1.3†
Hartford	0.5*	St. Paul	0.7	Indianapolis	1.3
St. Louis	0.5	Yonkers	0.7	Oklahoma City	1.3
Syracuse	0.5*	Louisville	0.8†	Albany	1.4
Worcester	0.5*	Norfolk	0.8	Baltimore	1.5†
Boston	0.6†	Atlanta	0.9†	Camden	1.6*
Cincinnati	0.6	Evansville	0.9*	Birmingham	1.7†
Long Beach	0.6	Jersey City	0.9	Chattanooga	1.7

Second Rank: from 2.0 to 4.9 (Thirteen Cities)

Tampa	2.0†	Fort Worth	2.7†	Miami	3.6†
San Antonio	2.3	Richmond	2.7†	Knoxville	3.9
Spokane	2.4†	Wilmington	2.7	Dallas	4.3†
Trenton	2.4†	Houston	3.1	Nashville	4.9†
		Memphis	3.1†		

Third Rank: from 5.0 to 5.9 (Three Cities)

Jacksonville	5.2
New Orleans	5.5†
El Paso	5.9

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.

excess of 1.0. Not only do these cities as a group have a low death rate but actually the lowest number of deaths. There were eleven deaths in 1938, compared with twenty-one in 1937. The Iowa State Health Department reports that no statistical correction is as yet available regarding deaths among nonresidents and consequently such information is not included in the

report for Des Moines. It is likely, however, that this same error might creep into the statistics of many other cities widely scattered throughout the United States.

The eight cities of the West South Central group did not fare so well. Here the rate increased from 2.34 in 1937 to 3.52 in 1938. The actual number of deaths increased from forty-nine to seventy-four. The death rate increased in six of the eight cities, Oklahoma City and San Antonio being the only two with a lower death rate. El Paso, with no death in 1937, reports a rate of 5.9 (six deaths) in 1938. All but one of these deaths are among residents. Oklahoma City records no data concerning deaths among nonresidents. Of six deaths in San Antonio, four were among residents; of eleven in Houston, nine were among residents; of twenty-nine in New Orleans, fourteen were among residents. With no death in a resident in 1936 and 1937, Dallas reports thirteen deaths for 1938, of which nine were among nonresidents.

TABLE 11.—*Number of Cities with Various Typhoid Death Rates*

	No. of Cities	10.0 and Over	5.0 to 9.9	2.0 to 4.9	1.0 to 1.9	0.1 to 0.9	0.0
1906-1910	77	75	2	0	0	0	0
1911-1915	79	58	19	2	0	0	0
1916-1920	84	22	32	30	0	0	0
1921-1925	89	12	17	48	12	0	0
1926-1930	92	3	10	30	37	12	0
1931-1935	93	0	6	17	28	42	0
1930	93	2	6	30	23	22	10
1931	93	2	6	23	28	22	12
1932	93	1	7	13	29	29	14
1933	93	0	7	18	19	33	16
1934	93	0	9	11	27	23	23
1935	93	0	7	15	18	29	24
1936	93	0	3	15	21	36	18
1937	93	0	1	13	26	26	27
1938	93	0	3	13	14	34	29

TABLE 12.—*Total Typhoid Rate for Seventy-Eight Cities 1910-1938* *

	Population	Typhoid Deaths	Typhoid Death Rate per 100,000
1910	22,573,435	4,637	20.54
1911	23,211,341	3,950	17.02
1912	23,835,399	3,132	13.14
1913	24,457,989	3,285	13.43
1914	25,091,112	2,781	11.08
1915	25,713,346	2,434	9.47
1916	26,257,550	2,191	8.34
1917	26,865,408	2,016	7.50
1918	27,086,696†	1,824†	6.73
1919	27,735,083†	1,151†	4.15
1920	28,244,878	1,088	3 85
1921	28,859,062	1,141	3.95
1922	29,473,246	963	3.26
1923	30,087,430	950	3.16
1924	30,701,614	943	3.07
1925	31,315,598	1,079	3.44
1926	31,929,782	907	2.84
1927	32,543,966	648	1.99
1928	33,158,150	628	1.89
1929	33,772,334	537	1.59
1930	34,386,717	554	1.61
1931	35,137,915	563	1.60
1932	35,691,815	442	1.24
1933	35,691,815	423	1.18
1934	35,401,715	413	1.17
1935	35,401,715	348	0.98‡
1936	36,216,404	336	0.93§
1937	36,771,787	280	0 76#
1938	36,972,985	248	0.67##

* The following fifteen cities are omitted from this table because data for the full period are not available: Canton, Chattanooga, Dallas, Fort Wayne, Jacksonville, Knoxville, Long Beach, Miami, Oklahoma City, South Bend, Tampa, Tulsa, Utica, Wichita, Wilmington.

† Data for Fort Worth lacking.

‡ The rate for ninety-three cities in 1935 was 1.03 (total population 37,437,812, typhoid deaths 385), whereas in 1930 it was 1.64, and in 1933 and 1934 it was 1.24 and 1.25, respectively. The 1931-1935 average for the ninety-three cities is 1.31.

§ Rate for ninety-three cities in 1936 was 0.96 (total population 38,249,094, typhoid deaths 365).

Rate for ninety-three cities in 1937 was 0.82 (total population 38,885,435, typhoid deaths 318).

Rate for ninety-three cities in 1938 was 0.74 (total population 39,143,556, typhoid deaths 291).

Special Note.—Deaths for 1936 have been corrected, as Yonkers originally reported seven deaths and later corrected report to one death.

The cities in the Mountain and Pacific states show a continued reduction in the rate (0.52 in 1938, 0.68 in 1937). There were recorded twenty-two deaths in 1938, twenty-eight deaths in 1937. In 1938 there were five cities with no death (Seattle, Tacoma, Portland, Salt Lake City, San Diego). Three additional cities (Oakland, Los Angeles, Spokane) record one third or more deaths which were in nonresidents. There was but one city with a rate in excess of 2.0 (Spokane). There were but three deaths in Spokane, two of which were among nonresidents. Two cities (Seattle, Tacoma) report no typhoid death in 1937 and 1938. Of five deaths in San Francisco, four were among residents. Of eight deaths in Los Angeles, five were among residents. Denver does not keep a record of deaths among nonresidents.

THE HONOR ROLL

The number of cities with no death from typhoid has increased to twenty-nine. In 1936 there were but eighteen such cities, in 1937 twenty-seven. Of particular significance are the data in table 9, which furnishes the names of thirteen cities with no typhoid death in 1937 and 1938. Bridgeport and Fort Wayne have continued their excellent records. While last year there was but one city in the third rank (Miami), and this city would not have been included in this rank had the correct number of deaths been recorded, in 1938 we find three cities in this group, only one of which reports a significant number of deaths among nonresidents. As already stated, several of the cities in the first rank would appear in the honor roll were they not charged with deaths in nonresidents. Table 11 continues to show a swing "to the right," although the number of cities in the group having death rates from 5.0 to 9.9 has increased from one to three.

TABLE 13.—*Total Typhoid Death Rate per Hundred Thousand of Population for Ninety-Three Cities According to Geographic Divisions*

	Population	Typhoid Deaths		Typhoid Death Rates				
		1938	1937	1938	1937	1931-1935	1926-1930	1925
New England	2,645,438	12	12	0.45	0.45	0.70	1.31	2.48
Middle Atlantic	13,469,996	59	68	0.44	0.51	0.80	1.40	2.97
South Atlantic	2,638,626	46	51	1.74	1.96	2.70	4.50	7.01*
East North Central	9,907,440	35	61	0.35	0.62	0.75	1.29†	2.32†
East South Central	1,355,080	32	28	2.36	2.10	4.81	8.31	13.00
West North Central	2,785,688	11	21	0.40	0.76	1.24	1.83	3.43
West South Central	2,120,553	74	49	3.52	2.34	5.36	7.32‡	13.08§
Mountain and Pacific	4,220,735	22	28	0.52	0.68	0.88	1.80	2.33

* Lacks data for Jacksonville and Miami.

† Data for South Bend for 1925-1929 are not available.

‡ Lacks data for Oklahoma City in 1926.

§ Lacks data for Oklahoma City.

NEW LOW RECORDS ATTAINED

For the seventy-eight cities (table 12) for which complete data are available since 1910, there occurred 248 deaths from typhoid in 1938, which is the lowest of record (280 in 1937, 336 in 1936). The rate for this group of cities is for the fourth consecutive year less than 1.0. The rate for the ninety-three cities studied in 1938 is also below 1.0 (0.74) and below the corresponding rate for 1937 (0.82). This annual review again showed a downward trend in the death rate from

typhoid in the large cities of the United States. No outbreak of epidemic proportion has been recorded in these cities. Routine vaccination of the population is not practiced except under flood conditions. However, in progressive communities vaccination is urged for contacts to cases and for persons who travel widely in countries where sanitary conditions are not of the best. There seems to be a trend in some places to encourage the inoculation of foodhandlers, especially in the Southern states. The noteworthy gains for 1938 have been made in the East North Central and West North Central cities. The New England and Middle Atlantic groups have, on the whole, maintained their excellent rates of many years' standing.

Council on Physical Therapy

THE COUNCIL ON PHYSICAL THERAPY HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORT.

HOWARD A. CARTER, Secretary.

E & J RESUSCITATOR AND INHALATOR ACCEPTABLE

Manufacturer: E & J Manufacturing Company, 139 South Verdugo Road, Glendale, Calif.

The E & J Resuscitator and Inhalator may be used for producing artificial respiration by mechanical means or simply as an inhalator for providing a concentration of oxygen or a mixture of oxygen and carbon dioxide around the mouth and nostrils. The apparatus is available in three models, one portable and two for hospital service. The portable unit weighs 100 pounds complete with gas cylinders. The resuscitator side[1] of the appliance produces artificial respiration by setting up in the lungs a sequence of alternating positive and negative pressures. The stroke automatically adjusts itself within limits to the lung capacity of the patient. While both positive and negative pressures are set at the factory, the gas flow may be regulated by a control valve at the will of the operator. Since the gas volume is adjusted to the lung capacity of the adult to produce the proper number of respirations per minute, the same flow would cause a more rapid respiratory rate in a child with smaller lung capacity. A regulator control valve is placed on the apparatus, which enables the operator to reduce gradually the flow of oxygen, thus automatically reducing the number of respirations per minute.

E & J Resuscitator and Inhalator.

The inhalator side[2] of the apparatus supplies a constant flow of oxygen or oxygen and carbon dioxide mixture to the mouth and nostrils. The transfer from the resuscitator to the inhalator side of the device is accomplished by means of a lever, so that either one or the other technic is independently available.

The E & J Resuscitator and Inhalator is designed to be used in emergencies in which natural respiration has failed with resulting asphyxia. The portable unit may be employed by fire and police departments, by life guards and mine rescue squads, and by other trained organizations rendering first aid. The hospital unit is provided for such respiratory emergencies as arise in hospital practice.

When used as a resuscitator, gas pressure in the tank operates the pistons contained within the mechanism. A face mask is fitted tightly over the patient's nose and mouth after an "Airway" (a wire device for holding down the tongue to clear a passage to the trachea) is placed in the patient's mouth. Gas, after passing through and actuating the apparatus, enters the face mask through a hose and is carried into the trachea and lungs at a pressure of from 12 to 14 mm. of mercury. There is a safety valve on the mask which is set at the factory and releases at approximately 18 mm. of mercury. The gas is withdrawn under a smaller negative pressure of from 7 to 9 mm. of mercury below the atmospheric level. The exhaled gas returns through another hose to a separate cylinder, creating the vacuum in the mechanism, and is finally discharged into the atmosphere. Briefly, therefore, the resuscitator is operated by the energy of the gas stored in the tank under pressure, while the selfsame gas is used in the lungs for resuscitation purposes. If the throat is filled with mucus, an aspirator, a separate accessory, is available which utilizes the negative pressure phase of the resuscitator cycle to clear the air passages.

After natural breathing has resumed, by the turn of a lever the respirator is turned off and the inhalator turned on for such time as is deemed necessary. When the inhalator side is used, the gas passes from the tank through a regulating valve, thence into a rubber breathing bag and finally to the patient's mask. On exhalation, it passes through the auxiliary exhalation valve and out to the atmosphere. The inhalator side of the apparatus is similar in operation to other inhalators accepted by the Council.

In cleaning the apparatus, the firm recommends the use of 20 per cent aqueous solution of green soap, worked into a good lather, and the face cushion, celluloid mask, mask valve and hose leading to the apparatus should be scrubbed with this solution. Then the parts should be rinsed with clear water, and given a second washing with an aqueous solution 50 per cent alcohol and allowed to dry.

The evidence that has been made available indicates quite clearly that the machine has demonstrated its worth in trained hands. The critical data consisted of many reports on the use of the resuscitator in the fields of surgery and obstetrics.

It must be pointed out, however, that the process of blowing oxygen into the lungs and aspirating it has a reverse effect on the circulation from that of normal respiration. Normal respiration tends to facilitate the exchange of gases in the lung capillaries. When the lung is inflated, even at very moderate pressures, circulation through the lung capillaries is retarded for an instant, since the blood pressure in these capillaries is very small. Stoppage here necessarily affects the entire circulation. However, periodic stops last only a moment, since the peak pressure (14 mm. of mercury) is maintained for only a short part of the cycle.

Another point should be borne in mind. While artificial respiration has been maintained by systems of this type in physiologic laboratories where experiments are performed on animals with the chest opened, the pressure required for adequate ventilation in such cases is much less than that required to distend the lung when the diaphragm must be forced down against the pressure of the abdominal contents and the bony thorax distended by pressure exerted by the lung from within.

In accepting the apparatus the Council wishes to stress two points, namely: (*a*) no artificial resuscitator or inhalator should replace the training of medical men and first aid men in the prone pressure method of resuscitation; (*b*) it is very important that this machine be used by well trained operators who have received their instruction from competent physicians.

The acceptance of this apparatus, therefore, is not to be regarded as a recommendation to abandon prone pressure methods of artificial respiration. In cases needing resuscitation the prone pressure method should be instituted immediately pending the arrival of any type of resuscitator, inhalator, or respirator, providing the patient's condition permits.

It is manifestly impossible to apply prone pressure artificial respiration in all conditions which arise in medical practice, as instanced in surgical operation on the abdomen when respiration fails. In such cases a mechanical device for maintaining artificial respiration over short periods may be very desirable. Operators who are trained to use the mechanical resuscitator should also be equally well trained in the prone pressure method of artificial respiration.

In view of its findings, the Council on Physical Therapy has voted to accept the E & J Resuscitator and Inhalator for inclu-

1. Resuscitator, an apparatus for revivifying, reviving or restoring from apparent death or from unconsciousness.
2. Inhalator, an apparatus for facilitating the breathing or drawing in of gas or a spray.

sion in its list of accepted apparatus, with an understanding of the limitations and hazards of positive pressure resuscitators, with the expectation that it will be used only by professional or other adequately trained personnel.

RESUSCITATION

A Comment by the Secretary of the Council on Physical Therapy

Historical documents record that man has tried innumerable methods, in one form or another, of reviving victims of asphyxia. Probably the earliest record of an attempt to resuscitate is in the Bible. Here it is said that Elisha brought back to life the son of a Shunamite woman by breathing into the mouth of the child.

We find also that in the early ages men employed burning and cutting of the skin, used boiling water, burning oil, whipping and noise as means of reviving the victim, as well as less drastic procedures, such as the application of heat by warm clay or ashes and warm poultices.[1] Counter shock, contrast baths, electrical stimulation, ducking in water, and blows on the soles of the feet and at the region of the heart are recorded. Two unusual methods were the drawing of a couple of teeth and the administration of wine or brandy. Of these two, if the patient was lucky enough to be revived, undoubtedly he preferred the latter rather than the former method. Fumigation has been used, that is, blowing tobacco smoke or charcoal fumes into the mouth or into the rectum. The rolling methods were tried; for example, rolling the patient over and over like a barrel, rolling him in a barrel or rolling him on a barrel. Manipulation, such as moving the arms in and out or up and down, working the legs, applying pressure alternately to the stomach and then to the chest or turning the victim back and forth from side to back are also recorded. Other methods were laying the patient face down or on his back and lifting his hips up and down, and countless other methods. Such were some of the practices of medical antiquity.

One method, placing the patient in a prone position and, among other operations, applying pressure to the small of the back, has survived. It is known as the Schafer prone pressure method. Careful investigation of the physiology involved has led to its acceptance and recognition.

When oxygen was discovered, it was used for resuscitation. At the present time a mixture of oxygen and carbon dioxide gas is an acceptable method, since investigation has shown that carbon dioxide stimulates the respiratory centers.[2]

Concurrently with some of the aforementioned methods, mechanical means were introduced in the sixteenth century. So we learn that Paracelsus of Switzerland in 1530 was the first to make use of the common fireside bellows for introducing air into the lungs of the apparently drowned person. In 1776 John Hunter improved the bellows for filling the lungs with fresh air and withdrawing exhaled breath from the patient. This method appeared to be so useful in the opinion of the Royal Humane Society of London that it was adopted by that organization in 1782. In 1911 the pulmotor appeared on the market. This was an automatic pumping device energized by the compressed gas within the tanks, which introduced gas into the air passages and sucked it out.

The report of the Council on Physical Therapy in this issue on the E & J Resuscitator and Inhalator precedes this item. It is energized by compressed gas stored in the two tanks in the carrying case. By a positive and negative pressure created at the mouth and nose within a face mask, gas passes back and forth within the air passages. The device automatically regulates itself to the size of the lungs and is provided with safety appliances. For the past seven years the Council on Physical Therapy has given careful consideration to mechanical resuscitating apparatus and has accepted the Drinker and Emerson respirators, both adult and infant size. The Council has also considered and accepted inhalators of various makes which provide an atmosphere of oxygen and carbon dioxide at the nose and mouth of the victim. These appliances, such as the H-H Inhalator and the Davis and the B-K Inhalators, are used in conjunction with the Schafer prone pressure method of resuscitation.

There follows a communication from Dr. Yandell Henderson and the Council's reply. Dr. Henderson is a recognized authority in the field of resuscitation and is a champion of the Schafer prone pressure method of resuscitation and one of the originators of the H-H Inhalator. He has opposed mechanical methods of the blow and suck type of resuscitator. One of his reasons is that the promotion of this kind of resuscitation will have a tendency to depreciate the value of the Schafer prone pressure method. In accepting the E & J Resuscitator the Council points out most emphatically that acceptance is not a recommendation to abandon the Schafer prone pressure method but that safety organizations, Boy Scouts, Red Cross and all other first aid groups should be carefully instructed in the use of this valuable measure. It is very important, the Council states, that all resuscitators and inhalators be used by well trained operators who have received their instructions from competent physicians.

As an indication of the care which the Councils of the Association take in consideration of products, the E & J Resuscitator is an excellent example. The Council on Physical Therapy has considered this product for a period of several years. During this time there have been numerous charges and countercharges received from several sources and in most cases the arguments were sincere. In such a situation the Council is not to be deterred from its cardinal principle—that it will not accept an apparatus for inclusion in its list of accepted apparatus until it is satisfied by the evidence and unless the manufacturer limits its claims to what the Council considers true and justified. The Council has spent a great deal of time in going over painstakingly the evidence which has been presented, which involves mechanical construction, principles, physiology and respiration, and other material.

1. Fisher, Hart Ellis: The Evolution of Resuscitation, Transactions of National Safety Council, 1931.
2. Henderson, Yandell: Resuscitation, J. A. M. A. **103**: 750 (Sept. 8), 834 (Sept. 15) 1934.

AN OPEN LETTER BY DR. YANDELL HENDERSON

To the Board of Trustees:—For some years past the American Medical Association has maintained a Council on Physical Therapy. Like the Council on Pharmacy and Chemistry, it is intended to afford the medical profession the technical advice that will maintain high standards of therapy and protect the public against harmful materials and methods. Yet in one of the most important fields of physical therapy—that of resuscitation—the influence of the Council, instead of being beneficial, is becoming extremely harmful. It is lowering standards and promoting methods and devices that will severely decrease the saving of life.

Three points require particular attention: 1. The Council on Physical Therapy is antagonizing a policy for general training in manual artificial respiration followed with marked success for many years by the American Red Cross. 2. It is approving apparatus that, if generally adopted, will seriously decrease the efficiency of the treatment of carbon monoxide poisoning and thereby largely increase the mortality. 3. It is tolerating and thereby virtually approving and promoting apparatus of the pulmotor type for application to the asphyxial newborn, which apparatus, if it works at all, administers a form of artificial respiration that tends rather to induce than to overcome atelectasis, and that in addition sometimes causes serious damage to the lungs.

I. Although I have no official authority to speak for the Red Cross, I have a reason for defending its methods of resuscitation, which I believe to be equally valid. Twenty-six years ago, in 1912, at the request of the director of the United States Bureau of Mines, the American Medical Association appointed a committee to investigate the subject of resuscitation from asphyxia by mine gases and, in particular, to recommend the best method of administering artificial respiration. The committee (generally referred to as the First Resuscitation Commission) included W. B. Cannon, chairman, G. W. Crile, Joseph Erlanger, S. J. Meltzer and myself. At the request of the National Electric Light Association this committee was enlarged by the addition of several electrical experts, including Elihu Thompson and A. E. Kennelly, to form the Second Resuscita-

tion Commission for the investigation of electric shock. Under both commissions we made very thorough investigations—among which I dealt particularly with manual artificial respiration; and on these investigations we based positive recommendations. These recommendations were at once adopted by the Bureau of Mines (Technical Paper 77, 1914), the United States Navy, the electric light industry and, most important of all, the American Red Cross. The chief recommendations were (1) the elimination of all mechanical appliances for giving artificial respiration under field conditions, and (2) universal training in the Schafer prone pressure method of artificial respiration. For the elimination of mechanical apparatus, of which four types were tested, several reasons were given: 1. At best such apparatus was found to be no more effective than the manual method. 2. A face mask frequently leaks and automatic apparatus often fails to operate as intended. 3. Experience proves that in any community where there is such apparatus the training in manual artificial respiration is neglected, the apparatus is sent for and the early moments of asphyxia, which are critical for the saving of life, are lost. 4. The form of respiration induced by the pulmotor, the appliance to which we devoted most careful consideration, was found to be distinctly unphysiologic and prone to induce atelectasis and to cause serious injury to the lungs.

In 1917 a third commission reviewed and generally confirmed these recommendations (*Science* **48**:563, 1918). Among its members were S. J. Meltzer, chairman, E. F. Du Bois, D. L. Edsall, W. H. Howell, Reid Hunt, C. A. Lauffer, J. W. Schereschewsky, G. N. Stewart, E. B. Vedder, C. H. Frazier and Yandell Henderson, and for the electrical industry Elihu Thompson, A. E. Kennelly, Wills Maclachlan and C. B. Scott. And in 1922 a fourth commission (*J. Indust. Hyg.* **6**:125, 1923), which included C. K. Drinker, chairman, W. B. Cannon, D. L. Edsall, H. W. Haggard, L. J. Henderson, Yandell Henderson, W. K. Lewis, F. W. Peabody and R. R. Sayers also disapproved mechanical artificial resuscitation devices, particularly those of the pulmotor type, and advocated general training in manual artificial respiration for use in such emergencies as drowning and electric shock. It was under this fourth commission that the treatment of carbon monoxide asphyxia by inhalation of a mixture of carbon dioxide with oxygen was developed and introduced (Drinker, C. K.: Carbon Monoxide Asphyxia, Oxford University Press, 1938. Henderson, Yandell: Adventures in Respiration, Baltimore: Williams & Wilkins Company, 1938).

Largely under the influence of the Red Cross, based on the reports of these commissions, more than 13 million policemen, firemen, boy scouts and others have been trained to administer manual artificial respiration and many lives are now thereby saved. It is deplorable that the influence of the Council on Physical Therapy should now be exerted against the recommendations of these commissions and against such training. Yet this influence is becoming effective, for a letter recently received asks me whether I advocate "resuscitators," by which name devices of the pulmotor type are now called, and notes "a strong tendency toward the use of resuscitators and elimination of the prone pressure method."

II. Having myself formerly served as a member of the Council on Physical Therapy, and having from time to time corresponded with its Chairman and Secretary, I have been led to conclude that the attitude of the Council in regard to the recommendations of these four commissions is not really one of opposition. It is merely one of ignorance. The Council is evidently unacquainted with the work that was done under these commissions. None of the members of the Council are specialists in the subject of resuscitation nor do they seem to realize that resuscitation is a very specialized subject. Each device that is submitted to the Council is merely referred to a referee; and while I was in the Council the reports of these referees, of whose names I was never informed, showed that their anonymous authors in many cases lacked both the general knowledge and the practical experience that the evaluation of any device in the field of resuscitation requires.

I will instance two such devices. One is an inhalator for the treatment of carbon monoxide asphyxia. When I wrote to the Secretary of the Council in regard to this device he replied "The Council has accepted the S. O. S. Inhalator put out by the Oxygen Equipment and Service Company of Chicago. This device, as you know, looks very much like other inhalators that have been accepted by the Council." Yet, in fact, this device is essentially different from and inferior to the inhalators now used by the rescue crews of city fire departments and other organizations. Those inhalators are particularly designed to administer a mixture of carbon dioxide with oxygen and to promote the rapid elimination of carbon monoxide by the avoidance of all rebreathing. This inhalator, on the contrary, can use only pure oxygen, for if carbon dioxide were added the small supply of oxygen in its little cylinders would be almost immediately exhausted. And it expressly provides for at least partial rebreathing, which will correspondingly prevent the elimination of carbon monoxide and prolong the asphyxia. It is, in fact, simply an oxygen inhalator like those used twenty years ago, before the modern treatment of asphyxia was developed.

The referee on this device was evidently unaware of the developments of recent years in regard to asphyxia and its treatment. And the Council in accepting his report either showed a similar degree of unawareness or else neglected to give the matter any serious consideration. The result of the approval of this device will be the loss of many lives that could and should be saved.

I venture therefore to suggest that, if the Council cannot devote the requisite knowledge, care and time to the examination of devices in the field of resuscitation, it could at least follow the lead and adopt the recommendations of another organization, namely the committee on safety of the American Gas Association. That association includes practically all of the city gas companies and is therefore vitally interested in holding the mortality from carbon monoxide asphyxia to a minimum. Its committee on safety does apply modern knowledge to this field and does devote the requisite time and care to its recommendations to afford the public the protection that it needs.

The other device to which I refer is the E & J Resuscitator. This device has been before the Council for about five years during which time its sales agents have continually claimed that it was about to be approved. And as two advertisements of it have appeared in THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION (a full page advertisement on June 25, 1938, and previously a smaller one on May 7), and as it has been exhibited at the annual meeting of the Association, a formal approval would scarcely have been more effective in foisting this device on hospitals, bathing beaches and various other agencies concerned with resuscitation.

Yet this so-called "resuscitator" is to all intents and purposes simply a pulmotor under another name. All the objections that applied to the pulmotor apply equally to this device, and it is needless to rehearse them. But one additional objection must be mentioned. It arises from the interest that has developed in recent years in regard to the asphyxia and resuscitation of the newborn. The lungs at birth are in a state of atelectasis, and even in the normal child they are only gradually dilated. It should be obvious then that in the treatment of asphyxia of the newborn nothing should be done to collapse them. Yet in the artificial respiration produced by this "resuscitator" and similar apparatus of the pulmotor type not only inflation but also deflation is applied. Its action, as should be expected, tends therefore not to promote but rather to oppose the dilatation of the lungs and often causes serious injury, demonstrable at autopsy. After application of this apparatus the survival of the baby is in fact rare. How injurious the E & J Resuscitator may be is shown in a recent paper by Dr. F. N. Coryllos. Dr. Coryllos was himself a supporter of the value of this device for use in connection with thoracic surgical operations. Nevertheless he found that, when healthy dogs were submitted to artificial respiration by means of it, the lungs after a few hours were in several cases too far collapsed and damaged for the animals to survive (*Surg., Gynec. & Obst.* **66**:698 [April] 1938).

The case against this device could scarcely be clearer or stronger. Yet year after year goes by and the Council still hesitates to approve or disapprove while the device appears to have an increasing sale at bathing beaches and in the maternity wards of hospitals.

III. If these two devices stood by themselves, the case against them would scarcely justify so unusual a motion as this appeal over the heads of the Council on Physical Therapy to the Board of Trustees of the American Medical Association. But in fact there is probably no subject other than that of perpetual motion apparatus that exercises so strong a lure for ignorant inventors as that of restoring the dead to life. There is certain to be in future, as in the past, a long succession of devices invented for this purpose. And it is of literally vital importance that, if there is to be a Council on Physical Therapy and it is to pass on such devices, it should be competent and use competent means and agents. In particular, it should free itself from the popular but fallacious assumption on which the invention of all pulmotors, resuscitators and other artificial respiration devices is based that the restoration of respiration in a nonbreathing patient is like starting the motor of an automobile: in other words, that if the thorax is put through the movements of breathing by vigorous "cranking" it will pick up the rhythm and continue to run. In fact, on the contrary, natural respiration is induced only by chemical not mechanical conditions; and those conditions are the supply of oxygen and carbon dioxide through the blood from the lungs to the respiratory center in the base of the brain. It is for this reason that the inhalators used by the rescue crews as adjuncts to manual artificial respiration on cases of submersion and electric shock are superior to all apparatus of the pulmotor type. The fact that such apparatus is able to inflate and deflate a rubber doll is impressive only to those who are untrained in the physiology of respiration.

YANDELL HENDERSON, PH.D., New Haven, Conn.

THE COUNCIL ON PHYSICAL THERAPY VOTED TO ADOPT THE FOLLOWING REPLY.

HOWARD A. CARTER, Secretary.

REPLY TO THE OPEN LETTER FROM DR. YANDELL HENDERSON

The Council on Physical Therapy has devoted much time to the entire subject of resuscitation in relation to all types of respirators, resuscitators and inhalators.

Dr. Henderson's first charge is that the Council on Physical Therapy "is lowering standards and promoting methods and devices that will severely decrease the saving of life." The Council has on many occasions emphasized the importance of the Schafer prone pressure method of artificial respiration. In the booklet "Apparatus Accepted" the Council describes and promotes this life-saving measure. The Council will continue to publicize this important method and will vigorously combat any effort on the part of an institution or organization to discard it.

Dr. Henderson's second charge is that the Council "is approving apparatus that, if generally adopted, will seriously decrease the efficiency of the treatment of carbon monoxide poisoning and thereby largely increase the mortality." The Council has accepted the Drinker and Emerson types of respirators, which are of unquestionable value especially in infantile paralysis: the H-H Inhalator, the B-K Inhalator, and the Davis Inhalator, which functions on the same principle as the H-H Inhalator. All of these appliances have been on the Council's accepted list for several years.

Further on in the letter Dr. Henderson refers to the S. O. S. Inhalator. Although the Council has given consideration to this product, it has not released a report of acceptance on this unit and does not expect to until further evidence is submitted by the manufacturer to substantiate certain claims made. At present, therefore, this product stands neither accepted nor rejected. The firm has been asked to submit further evidence of its efficacy as, for example, a report of animal experiments definitely showing the amount of carbon monoxide present in the bag when the inhalator is being used in resuscitation.

The third charge is that the Council "is tolerating and thereby virtually approving and promoting apparatus of the pulmotor type for application to the asphyxial newborn, which apparatus, if it works at all, administers a form of artificial respiration that tends rather to induce than to overcome atelectasis, and that in addition sometimes causes serious damage to the lungs." Following this charge Dr. Henderson gives a detailed study of appointments of commissions by the Red Cross, American Medical Association, National Electric Light Association and engineering societies and a report of the findings of these commissions. He intimates that the Council is not familiar with the work of these commissions or their results and definitely states that the Council has no member or members familiar with the subject of resuscitation—"nor do they seem to realize that resuscitation is a very specialized subject." He further condemns the Council's method of referring these appliances to referees "of whose names I was never informed. . . ."

In reply the Council would state that it has made exhaustive studies of the E & J Resuscitator (the product to which Dr. Henderson refers later on in his letter) to satisfy itself that the apparatus was not of the old type pulmotor. The Council is thoroughly familiar with the work of the commissions to which Dr. Henderson refers and also with their reports concerning the Schafer prone pressure method of resuscitation, to the use of the inhalator type of apparatus and to the condemnation of the old type pulmotor.

Investigation indicates that the S. O. S. Inhalator has been used by several fire and police departments in the vicinity of Chicago with success. This product, therefore, is definitely a product in competition with accepted inhalators.

Furthermore, the Oxygen Equipment and Manufacturing Company is refilling tanks with carbon dioxide and oxygen mixture, which is the mixture furnished by the Mine Safety Appliances and marketed under the trade name of "Carbogen." (Carbogen is not accepted by the Council on Pharmacy and Chemistry.) The refilling of tanks has reduced the cost of this mixture, especially in the vicinity of Chicago. Furthermore, one of the charges of Dr. Henderson, that is, that the tanks are too small and that "its little cylinders would be almost immediately exhausted," is denied by the manufacturer, since the tanks are one-half the size of those furnished by the Mine Safety Appliances Company and other firms. The firm points out that a user of this equipment can lay in a supply of tanks and can, in fact, connect the unit to a large sized oxygen tank if the occasion arises.

The E & J Resuscitator and Inhalator was first discussed in Queries and Minor Notes in THE JOURNAL Nov. 16, 1929, page 1583. The reply condemned the E & J Resuscitator. In February 1934 the E & J Resuscitator and Inhalator was submitted to the Council for consideration. After investigation, a report of rejection was referred to the firm. The E & J Manufacturing Company pointed out that in its opinion the report was in error and submitted arguments that convinced the majority of the Council members that further investigation was indicated.

A leading chief surgeon of a large corporation, who had considerable experience with the E & J Resuscitator and Inhalator, not only wrote to the Council but made a trip to Chicago to interview the Chairman, the Secretary and one other member of the Council. He presented evidence showing that a number of these resuscitators were in use in various plants of which he was in charge and that they had rendered definite life-saving service in many instances.

The Council has selected specialists of known reputation to serve as referees for all inhalators and resuscitators. Since 1935 at least four referees, specialists in the field, have investigated independently the E & J Resuscitator and Inhalator and made reports to the Council.

During the year 1936 the E & J Manufacturing Company submitted evidence to the Council accumulated by the late Dr. Pol Coryllos. Dr. Coryllos's paper was published in *Surgery, Gynecology and Obstetrics* in April 1938 but the substance of the report, together with motion pictures, was furnished the Council in 1936. The work of Dr. Coryllos, in the opinion of the Council, was well done and convinced the members that the E & J Resuscitator and Inhalator was a safe appliance, that it had real life-saving value and that an injustice would have been done to its manufacturer if the original report of rejection had been published.

Thirty-nine different physicians of recognized professional ability were asked by the Council to submit reports of cases treated with the E & J Resuscitator. The vast majority of these reports were highly favorable to the resuscitator in spite of the fact that many of the patients were practically moribund at the time when resuscitation was initiated.

Finally, the Council had submitted to it a report of Dr. D. Ben Martinez, published in THE JOURNAL Aug. 14, 1937, in which he reported 500 cases of resuscitation of the newborn; the E & J Resuscitator was used in these cases.

A large midwestern fire department uses the E & J Resuscitator and Inhalator in preference to inhalators, giving as reasons: 1. Inhalators require at least two, or preferably three, men to operate, while the E & J Resuscitator requires but one man. 2. Inhalators require more space to use than does the resuscitator. 3. The E & J Resuscitator is more practical when used on a pregnant woman or on a person with broken ribs. 4. Better results are obtained with the E & J Resuscitator. The fire department has defended its use of the E & J Resuscitator in the face of opposition. It has convinced its city's health department that the resuscitator is efficient. The department had three E & J Resuscitators in 1931 and in 1934 purchased seven more and now it has twelve. Squads are well trained in the use of this apparatus and are also well trained in the use of inhalators and the Schafer prone pressure method. They demonstrated completely their ability to use this apparatus to the complete satisfaction of a committee of the Council.

Therefore, in reply to Dr. Henderson's charges that the Council on Physical Therapy is not familiar with resuscitation, that it depends simply on the statements of referees of questionable ability, and that it is accepting apparatus that will "lower standards and promote methods and devices that will severely decrease the saving of life," the Council submits the foregoing information to demonstrate the thoroughness of its investigation of the E & J Resuscitator and the effort the Council has put forth to avoid the loss of a single life through any of its published actions.

COUNCIL ON PHYSICAL THERAPY.
HARRY E. MOCK, M.D., Chairman.
HOWARD A. CARTER, Secretary.

Council on Pharmacy and Chemistry

REPORT OF THE COUNCIL

THE COUNCIL HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORT.

PAUL NICHOLAS LEECH, Secretary.

THE PRESENT STATUS OF TESTOSTERONE PROPIONATE: THREE BRANDS, PERANDREN, ORETON AND NEO-HOMBREOL (ROCHE-ORGANON) NOT ACCEPTABLE FOR N. N. R.

Within the past few months extravagant claims for the action of the male sex hormone testosterone have appeared in professional and lay publications. The naturally popular appeal of this substance has aroused wide interest with the aid of ample newspaper publicity. It may eventually prove that this substance, testosterone, or its esters will be a valuable addition to our armamentarium of glandular therapy; but it is the Council's belief that many claims for it have been grossly exaggerated. The male hormone has only recently been made available for clinical use and there have already been widespread announcements of its remarkable effects, both physical and psychic, which are a long way from actually being established. In view of the uncontrolled advertising and publicity, the Council is issuing this report on the status of the male hormone.

Testosterone, the male or testicular hormone, was first isolated from bulls' testes. It is not to be confused with androsterone, which appears in human urine. After establishment of its formula, Ruzicka was able to prepare the identical substance synthetically. It is generally accepted that testosterone is the substance secreted by the interstitial cells of the testes and that its function is the induction and maintenance of the secondary sex characteristics of the male. Fatty acid esters of testosterone are more slowly absorbed from the site of injection than the free testosterone. It apparently acts by simulating gonadal activity and in this manner the physiologic response has been observed to improve considerably. The most widely used ester at present is the propionate of testosterone.

In the clinical use of most endocrine preparations it seems to be a fairly consistent rule that the response to any hormone is most efficient and satisfactory when there already exists a clearly defined deficiency of that substance. This rule holds quite well for the male hormone and aids in indicating the use of this substance. Therapy has been attempted in the following conditions and, while the treatment of all of them is still in the experimental stage, it appears that results are promising in only a few.

EUNUCHOIDISM

The most careful studies in the clinical use of testosterone have been made in cases of eunuchoidism.[1] The cases of testicular deficiency reported by these workers were well defined. The testes were either absent or atrophic or else retained in the abdomen. The penis was underdeveloped and the prostate hypoplastic. Erections were infrequent or feeble and libido was negligible. Other signs and symptoms were typical: deficiency of pubic, facial and body hair, fine boyish skin and high pitched voice. Psychic conflicts were frequent, rendering the patient anxious, depressed and apathetic with a feeling of social inferiority. Occasionally the patients suffered from hot flashes, irritability and depression, symptoms typical of the climacteric.

The response to testosterone propionate was reported to be excellent. Within a short time sexual desire was initiated, erections became frequent and satisfactory, and nocturnal emissions occurred. After several months of continuous treatment the hair growth became heavier, the skin reverted to the adult type and the voice changed to a lower pitch. The prostate in a few cases enlarged somewhat and the penis approached normal dimensions. The psychic responses were no less spectacular. Relief from climacteric distress was complete, self assurance returned and emotional stability was manifested. Some of the beneficial results on the psyche may have been merely the reflection of the patient's pleasure in the physical changes and the sensations of sexual activity.

The dosage required to bring about these changes varied considerably. According to Kenyon,[2] from 7 to 21 mg. of testosterone is metabolized and lost daily in the urine and on these grounds he believes that 25 mg. daily is a reasonable amount for injection. Results have been obtained with smaller doses, but the preponderance of opinion indicates that from 75 to 150 mg. per week is necessary for complete replacement therapy. It cannot be stressed too strongly that the desired effects persist only as long as the therapy is maintained. No cure should be expected and regression of all beneficial changes takes place soon after the last injection. It is obvious, therefore, that androgenic therapy is an expensive procedure. Although the experimental treatment of eunuchoidism has proved successful, social and economic factors are formidable obstacles when this therapy is applied to the private patient. The careful selection of cases and the use of great caution are demanded for the proper treatment of this condition.[2a]

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1. Hamilton, J. B.: Treatment of Sexual Underdevelopment with Synthetic Male Hormone Substance, Endocrinology **21**: 649 (Sept.) 1937. Vest, S. A., Jr., and Howard, J. E.: Clinical Experiments with the Use of Male Sex Hormone: Use of Testosterone Propionate in Hypogonadism, J. Urol. **40**: 154 (July) 1938. Kenyon.[2]

2. Kenyon, A. T.: The Effect of Testosterone Propionate on the Genitalia, Prostate, Secondary Sex Characters and Body Weight in Eunuchoidism, Endocrinology **23**: 121 (Aug.) 1938.

2a. A well known news weekly has recently given wide publicity to the method adopted by Foss in England of administering to patients testosterone propionate by inunction. Following this work, several firms have put on the market ointments containing testosterone propionate. Foss and also Parkes administered to several eunuchs doses of from 20 to 50 mg. of testosterone propionate in about 2 cc. of ointment daily in order to obtain satisfactory results. The products marketed in this country contain only 2 mg. of the androgen per cubic centimeter of ointment. It is apparent, therefore, that to administer the appropriate amount of active material the patient must anoint himself with from 10 to 25 cc. of ointment daily. This would, of course, make quite a mess.

IMPOTENCE

The great majority of young men who complain of impotence suffer from so-called psychic impotence. The few cases reported in the literature which were treated with androgens did not respond very favorably.[3] This is in line with the rule just stated that the male hormone works best when there exists a deficiency. The males with a psychic disturbance have normally functioning testes and the additional male hormone provided by injection does not appear to correct this disturbance. Transitory improvement reported in several cases is probably the result of suggestion. There is no evidence that testosterone propionate has an aphrodisiac action in males with normal testicular function.

SENILITY

It appears that many pharmaceutic firms would have one believe that in the ampule of testosterone propionate lies the Fountain of Youth. The few reports in the literature of attempted rejuvenation are not at all promising. Laroche and Bompard[4] injected several aged men with this material and failed to improve their sexual powers appreciably. Apparently the action of male hormone on the masculinity of an individual is effective only within a certain age limit. Other factors, nervous or circulatory, probably play a role in the mechanism of erection and their absence in elderly males may prevent the response to testosterone. Premature impotence may well fit into this category, but sufficient data are not at hand from which conclusions may be drawn. The involutional melancholia of males, for which testosterone has been suggested, has not been subjected to adequate trials to justify androgenic therapy other than on an experimental basis.

STERILITY

It is known that testosterone propionate will maintain spermatogenesis in the hypophysectomized rat, but once tubular degeneration has set in, it is unable to stimulate sperm formation. There is apparently some species difference in the response of the testes to androgens. Wells and Moore have shown that injections of male hormone stimulate spermatogenesis in the ground squirrel, while Moore and Price and others have demonstrated an atrophy of the testes in similarly treated adult rats. There is little clinical evidence that this substance is of any use in human male sterility. The few cases of therapeutic success reported in the literature certainly do not warrant the use of androgens in this disorder at the present time.

PROSTATISM

There are a number of reports dealing with the use of male hormone in prostatism.[5] The impression seems general that improvement is evidenced in most cases on this type of treatment. In long standing cases of complete or incomplete retention there was a return of spontaneous urination soon after the first few injections. Frequency and nocturia decreased. The amount of residual urine diminished and the general condition of the patient improved following amelioration of the distress of prostatism. In most cases of prostatic hypertrophy there was no decrease in size of the organ. Laroche and his co-workers[6] obtained in several cases a diminution in size of the prostate, but they were careful to point out that this was due probably to the disappearance of congestion and inflammation and not due to any regression of the prostatic adenoma. In this regard it seems ill advised to describe, as several authors do, the androgenic therapy as treatment of prostatic hypertrophy, when no evidence is at hand that the excess tissue is appreciably affected. As a matter of fact, it has been recently shown that "the injection of 285 to 1,125 mg. of testosterone propionate in from 12 to 95 days results in no significant restoration of the involuted prostate of presenility and in no observable alteration in the histological appearance of the tissues formed in benign hypertrophy."[7] The actual mode of action of the androgen is unknown, the improvements being due possibly to an improvement in sphincter action, or relaxation of spasm, an increase in bladder tone or an attenuation of prostatic congestion.

On the other hand, Clarke[8] believes that conservative treatment is just as effective. He has followed ninety-three cases of prostatism and shown a large number of satisfactory responses on instrumentation alone. He suggested that a period of five years is necessary before results of endocrine therapy can be reliably assessed. The view taken by Riches[9] is an admirable one and strikes a note of caution in accepting this newer treatment of prostatism:

> The natural history of prostatic obstruction is one of frequent remissions and relapses, and many vaunted remedies in the past have owed their ephemeral reputations to their use at a time when acute congestive retention has just been relieved by catheterization. After a period perhaps of years it has been found that the prostatic enlargement has steadily progressed and has ultimately required operation when the patient is older and less fit and his kidneys have begun to fail. Herein lies the greatest danger of treatment by male hormone and it must be emphasized that all such treatment is still in the experimental stage, and is, therefore, only justified in patients who are not so ill as to need active surgical treatment or are too ill to stand much chance of surviving such treatment. The field for experiment is thus greatly reduced and the assessment of results is made difficult by the normal remissions and relapses incident to the disease.

CRYPTORCHIDISM

Hamilton[10] has induced descent of the testes in immature monkeys by means of testosterone propionate injections. He has also obtained these results in cryptorchid boys and has suggested that androgens may be of value in the differential diagnosis of obstructive and nonobstructive cryptorchidism. By administering such therapy, the physician need not wait until puberty to know whether the testes will descend spontaneously. In Hamilton's opinion, male hormone may also aid in the surgical treatment of undescended testes. These recommendations are attractive, but approval must await further clinical trial.

On the other hand, Kunstadter[11] warns against the induction of precocious puberty in boys by means of androgens. He observed some patients who were relatively sensitive to the androgenic substance, developing definite masculine traits after the administration of a moderate amount of this material. He considered these reactions harmful to the patient, especially from the psychologic standpoint. There have been several cases reported in the literature in which boys on androgenic treatment have resorted to masturbation and other undesirable behavior. It is highly important to remember that normal puberty varies considerably in its time of appearance. The diagnosis of hypogonadism in boys should be made with reserve and the endocrine treatment on the presumption of this condition should be accordingly avoided.

GYNECOLOGIC CONDITIONS

Estrous cycles of rodents have been suppressed by means of testosterone administration on the basis of its similarity to progesterone. In the monkey, Zuckerman[12] inhibited menstruation and obtained involution of the endometrium and ovarian atrophy by injections of androgenic substance. Inhibition of menstruation has also been obtained in the human being by Hartman[13] and by Papanicolaou and his co-workers.[14] Several

3. Miller, N. E., and others: Mental and Behavioral Changes Following Male Hormone Treatment of Adult Castration, Hypogonadism and Psychic Impotence, Proc. Soc. Exper. Biol. & Med. **38:** 538 (May) 1938. Laroche and Bompard.[4]

4. Laroche, G., and Bompard, E.: Essais de traitement de l'impuissance sexuelle masculine par l'hormone male, Progrès méd. **66:** 530 (April 9) 1938.

5. Day, R. V., and others: Sex Hormone Therapy for Prostatism, Am. J. Surg. **39:** 100 (Jan.) 1938. Laroche and others.[6]

6. Laroche, G., and others: Prostatic Hypertrophy: Results of Therapy with Testosterone Acetate and Propionate, Presse méd. **45:** 932 (June 23) 1937.

7. Moore, R. A., and McLellan, A. M.: A Histological Study of the Effect of the Sex Hormones on the Human Prostate, J. Urol. **40:** 641 (Nov.) 1938.

8. Clarke, Ruscoe: Prostate and Endocrine—Control Series, Brit. J. Urol. **9:** 254 (Sept.) 1937.

9. Riches, E. W.: The Use of Male Sex Hormones, Practitioner **140:** 60 (Jan.) 1938.

10. Hamilton, J. B.: Effect of Male Hormones upon the Descent of the Testes, Anat. Rec. **70:** 533 (April 25) 1938.

11. Kunstadter, R. H.: Induction of Premature Puberty with Androgenic Substance, Endocrinology **23:** 661 (Nov.) 1938.

12. Zuckerman, S.: Inhibition of Menstruation and Ovulation by Means of Testosterone Propionate, Lancet **2:** 676 (Sept. 18) 1937.

13. Hartman, C. G.: Menstruation Inhibiting Action of Testosterone, Proc. Soc. Exper. Biol. & Med. **37:** 87 (Oct.) 1937.

14. Papanicolaou, G. N., and others: Inhibitory Effects of Male Sex Hormone on Human Menstruation and Their Evaluation by Vaginal Smears, Proc. Soc. Exper. Biol. & Med. **37:** 689 (Jan.) 1938.

investigators[15] have treated cases of functional menorrhagia with testosterone propionate and have been able to decrease the uterine bleeding. Hundreds of milligrams of the material was usually necessary to produce the desired results. Desmarest and Capitain[16] have reported beneficial results in mastopathy with somewhat smaller doses (100-200 mg.). Other conditions in which male hormone therapy has received trials are the menopause, undesired lactation and dysmenorrhea. Such treatment should be reserved for the present for the specialist in this field, since very little is known as to the proper handling of these cases except that to obtain desired results in most cases it appears necessary to employ tremendous doses of testosterone propionate. Very little is known regarding the permanence of these results. The practicability of this therapy is, therefore, rather doubtful just now. Furthermore, a definite harm is risked when male hormone is administered to females. Greenhill and Freed[17] have induced a significant degree of virilism in women treated with this substance for certain gynecologic disorders including hirsutism and low voice. Other undesirable reactions may eventually be demonstrated.

MISCELLANEOUS

There are other conditions for which androgenic therapy has been suggested, such as atrophic rhinitis, acne and gynecomastia. The available data in such cases are either absent or inadequate to justify any claims of usefulness.

CONCLUSIONS

According to our present knowledge, testosterone propionate shows promise in only a few conditions. Careful studies have shown that it is adequate replacement therapy in true testicular deficiency (eunuchoidism), but the applicability of this therapy in actual practice is still undetermined. Its employment in cases of cryptorchidism, though not well established clinically, is on a sound physiologic basis. The relief so often elicited by testosterone propionate in cases of urinary retention due to prostatism is quite gratifying, but a reliable evaluation of the benefits or dangers of this treatment must await extensive experimentation over a period of years. All other claims are either exaggerated or immature and should be disregarded until substantial evidence becomes available on which to evaluate them.

The Council deferred further consideration of testosterone propionate until it has been properly evaluated by ample clinical experience.

On the basis of the foregoing report and without a detailed investigation of the advertising policies of the firms which market this substance, the Council also declared the marketed brands of testosterone propionate, namely Perandren (Ciba Pharmaceutical Products, Inc.), Oreton (Schering Corp.) and Neo-Hombreol (Roche-Organon) (Hoffmann-LaRoche, Inc.), unacceptable for inclusion in New and Nonofficial Remedies.

NEW AND NONOFFICIAL REMEDIES

THE FOLLOWING ADDITIONAL ARTICLE HAS BEEN ACCEPTED AS CONFORMING TO THE RULES OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR ADMISSION TO NEW AND NONOFFICIAL REMEDIES. A COPY OF THE RULES ON WHICH THE COUNCIL BASES ITS ACTION WILL BE SENT ON APPLICATION.

PAUL NICHOLAS LEECH, Secretary

RACÉPHEDRINE HYDROCHLORIDE (See THE JOURNAL, April 1, 1939, p. 1257).

Racéphedrine Hydrochloride-Gane's Chemical Works, Inc.—A brand of racéphedrine hydrochloride-N. N. R.

Manufactured by Gane's Chemical Works, Inc., New York. No U. S. patent or trade mark.

15. Loeser, A. A.: Action of Testosterone Propionate on Uterus and Breast, Lancet **1**: 373 (Feb. 12) 1938. Foss, G. L.: Action of Testosterone Propionate on Female: Clinical Application and Dosage, Lancet **1**: 992 (April 30) 1938. Desmarest and Capitain.[16]
16. Desmarest and Mme. Capitain: Testosterone Acetate, Therapy of Mastopathies: Seventeen Cases, Presse méd. **45**: 777 (May 26) 1937.
17. Personal communication.

Council on Foods

ACCEPTED FOODS

THE FOLLOWING PRODUCTS HAVE BEEN ACCEPTED BY THE COUNCIL ON FOODS OF THE AMERICAN MEDICAL ASSOCIATION AND WILL BE LISTED IN THE BOOK OF ACCEPTED FOODS TO BE PUBLISHED.

FRANKLIN C. BING, Secretary.

CELLU BRAND LOGANBERRY JUICE

Distributor.—Chicago Dietetic Supply House, Inc., Chicago.

Description.—Canned loganberry juice packed without added sugar.

Manufacture.—Good quality loganberries on which no spray material has been used are thoroughly washed, sorted for spoilage and foreign matter, preheated and pressed through ordinary press cloth. The juice is run into cans which are sealed and heat processed.

Analysis (submitted by distributor).—Moisture 90.0%, total solids 10.0%, ash 0.4%, fat (ether extract) 1.0%, protein (N × 6.25) 0.2%, crude fiber 0.02%, carbohydrates other than crude fiber (by difference) 8.4%, invert sugar 8.3%, sucrose 0.1%.

Calories.—0.4 per gram; 11 per ounce.

RED & WHITE BRAND FLOUR (BLEACHED)

Distributor.—Slocum-Bergren Company, Minneapolis.

Manufacturer.—Russell-Miller Milling Company, Minneapolis.

Description.—A long patent, bleached flour milled from hard winter wheat.

Manufacture.—Hard winter wheat is cleaned, washed, dried, scoured, tempered and milled by essentially the same procedures as described in THE JOURNAL, June 18, 1932, page 2210. The flour is bleached with 13 Gm. of a mixture of benzoyl peroxide and calcium phosphate and 2 Gm. of nitrogen trichloride per barrel.

Analysis (submitted by manufacturer).—Moisture 13.5%, total solids 86.5%, ash 0.5%, fat (ether extract) 1.5%, protein (N × 5.7) 11.2%, sucrose 1.3%, reducing sugar as invert sugar 1.7%, crude fiber 1.0%, carbohydrates other than crude fiber (by difference) 72.3%.

Calories.—3.48 per gram; 99 per ounce.

MILLER'S BRAND SPAGHETTI, MACARONI AND MACARONI PRODUCTS

Manufacturer.—Miller's Food Products, Inc., Los Angeles.

Description.—Spaghetti and macaroni, of various sizes and shapes, prepared from durum wheat semolina and water.

Manufacture.—Durum semolina and water in formula proportions are mechanically mixed and kneaded. The resulting dough is forced through a pressing and cutting machine and the pieces are dried and packed.

Analysis (submitted by manufacturer).—Moisture 11.4%, total solids 88.6%, ash 0.6%, fat (ether extract) 0.3%, protein (N × 5.7) 12.4%, crude fiber 0.4%, carbohydrates other than crude fiber (by difference) 74.9%.

Calories.—3.52 per gram; 100 per ounce.

CRADLE BABY BRAND STRAINED AND SEEDLESS APRICOT JAM

Manufacturer.—Glaser, Crandell Company, Chicago.

Description.—Canned strained apricot jam sweetened with sucrose. The jam may contain small amounts of added acid or pectin or both.

Manufacture.—Canned California apricots which conform to the specifications of the manufacturer are softened and subsequently treated as described for Cradle Baby Brand Strained and Seedless Fig Jam (THE JOURNAL, March 18, 1939, p. 1071).

Analysis (submitted by manufacturer).—Moisture 30.4%, total solids 69.6%, water insoluble solids 0.9%, water soluble solids 68.7%, ash 0.4%, protein (N × 6.25) 0.4%, crude fiber 0.3%, carbohydrates other than crude fiber (by difference) 67.7%, acidity 0.8%.

Calories.—2.72 per gram; 77 per ounce.

THE CASE OF BRINKLEY VS. FISHBEIN

PROCEEDINGS OF A LIBEL SUIT BASED ON AN ARTICLE PUBLISHED IN *HYGEIA*

On March 22 in the District Court of the United States for the Western District of Texas began the trial of the case of *John R. Brinkley* v. *Morris Fishbein,* based on an article published in *Hygeia,* entitled "Modern Medical Charlatans." The judge was the Honorable Robert J. McMillan; the attorneys for the plaintiff, Messrs. Morriss & Morriss of San Antonio and Phil Foster of Del Rio; the defendant was represented by Messrs. Loesch, Scofield, Loesch & Burke of Chicago, Brooks, Napier, Brown & Matthews of San Antonio, and Boggess, LaCrosse & Lowrey of Del Rio, Texas.

The case attracted much attention, particularly in Del Rio, the home of Brinkley. The following statement by Millard Cope in the San Angelo (Texas) *Standard-Times* gives the setting of the stage:

"Inwardly, however, Del Rioans are hoping for a victory for Dr. Brinkley because they have not forgotten what he and his hospital meant to Del Rio when he performed his operations in Roswell Hotel here.

"He maintained a monthly pay roll estimated at between $17,000 and $20,000 before moving the hospital to Little Rock, Ark. That meant plenty to business in Del Rio.

"The manager of one of Del Rio's largest department stores said that firm lost a thousand dollars a month in accounts when the Brinkley hospital was moved. Postal receipts, so often used as a business barometer, reflected a decline when Dr. Brinkley's hospital was moved. In 1937, for instance, postal receipts aggregated $54,167.54. (That was the year he testified as having grossed $1,100,000.) Last year, however, with the Brinkley hospital in Little Rock, receipts dropped nearly a fifth to $44,399.22.

"SAYS DEL RIO SUFFERED

"'Del Rio suffered its greatest loss when Dr. Brinkley moved to Little Rock.' declares H. Lippe, whose picture and curio business, he says, has shown a heavy loss.

"Over at the Del Rio National Bank, J. A. Walker reminds that any city the size of Del Rio will suffer when it loses such a pay roll as Dr. Brinkley maintained. 'Of course, Del Rio was getting along before we ever heard of Dr. Brinkley, and we'll still go along, but it's obvious that the hospital is missed.'

"The Chamber of Commerce, through James C. Netts, secretary, admits there are some vacant houses and waiting hotel and tourist camp rooms, which were filled when Dr. Brinkley attracted thousands of persons to his hospital annually. Some of the tourist camps were built to accommodate the influx of oldsters.

"Obviously, the Roswell Hotel, used by Dr. Brinkley as his hospital, is missing the patients and guests, drawn magnet-like to its registration desk. F. H. Childs, the manager, isn't experiencing so many full houses as before.

"Wherever one goes in Del Rio, virtually the same story is given. What city of 10,000 wouldn't miss a $20,000 pay roll every month, plus spendings of thousands who came seeking a more potent sex life? . . ."

In his article Mr. Cope gives the following description of Dr. Brinkley:

"Del Rioans have a kindly feeling for Dr. Brinkley for a lot of reasons other than the pay roll he maintained when he operated the hospital here. He maintains his home in Del Rio, a palace sort of Spanish-type architecture centered in a 12 acre tract. It's prettier at night, because the two lighted fountains are turned on, a dozen colors changing all the time as lights are thrown on water squirting 30 feet into the air. One has no difficulty in knowing its owner. The name "Dr. Brinkley" is in large letters on the gates to the blue-painted concrete drives, and the name flashes in neon on the fountains at night.

"Some say he has as much as $200,000 invested in the place. Inside, visitors see furniture brought over from France, imported tapestries, carpets, and works of art. There is an $11,000 pipe organ. In one of the rooms is a collection of perfumes which he has collected as a hobby and which now is valued at more than $5,000. He collects a variety of things. He's just as likely to return from a world cruise with a collection of cameras as flamingoes, freak dogs or shotguns.

"The unusual Dr. Brinkley has a flair for the unusual.

"MAKES NUMEROUS DONATIONS

"He thought the high school should have a business course and donated the typewriters, desks and other equipment. He provides candy and fruit for every child in Del Rio at a Christmas party each season. When the Rotary Club sought to increase its student loan fund, he offered to match it, reaching into his pocket to display three $500 bills. He offered $25,000 cash if the city would cause a similar amount for a public library to be raised.

"Though he built radio station XERA at Villa Acuna, Mexico, across the Rio Grande, he now denies any ownership, explaining he sold his interest and buys time as any other advertiser who broadcasts.

"He is driven to the court trial each morning by Mrs. Brinkley in a long, specially built body Cadillac, its color as bright a red as any fire truck, on which the name "Dr. Brinkley" is inscribed in 13 places, from the hub caps to the trunk. She stops the car at the curb, they kiss, and he's met by a group of admirers as he walks toward the federal building.

"A FORTUNE IN DIAMONDS

"Nattily attired, his close-cropped Van Dyke neatly trimmed for his appearance in court, and with diamonds flashing on his fingers, a tie-clasp and in a lodge emblem across his vest, Dr. Brinkley reflects some of the wealth he has acquired. The diamond in the ring on one finger is 14 carats, he says. On the other hand is a smaller one. There's a line of big ones in the tie clasp worn above the top button of his vest. Some estimate he may have spent as high as $100,000 for them. He has bought diamonds for Mrs. Brinkley, too. . . .

"Transportation is not much of a problem for Dr. Brinkley. He goes by land, air or water. He literally has a fleet of high-powered automobiles. There is a Lockheed Electra monoplane, with pilot and co-pilot, a 12-passenger ship for which he paid $58,000 and then added more than $20,000 worth of extras.

"ADMIRAL OF THREE YACHTS

"He's an admiral in his own right, owning three yachts. A picture of Dr. Brinkley in his private admiral's uniform hangs in Editor Travelstead's office. It is autographed: 'To Warrior Travelstead from Fighting Admiral Brinkley.' He would have bought another yacht, but the $1,000,000 price, he thought, was a little steep. It was the vessel on which abdicated King Edward VIII and the former Wally Simpson honeymooned in the Mediterranean. He had an associate contact a London broker as to the price.

"Hearing of the plight of orchard owners in the Rio Grande Valley in not being able to dispose of their fruit, he decided to buy an orchard. He passed up dividends for a few years by setting out new, choice trees. Now, his output is contracted. The grapefruit Dr. Fishbein ate Saturday morning in the Roswell Hotel came from the Brinkley orchard. . . .

"Judge R. J. McMillan's oblong little court room in the federal building is filled an hour before each day's trial begins, but that doesn't mean there is much of a crowd on hand. Its seating capacity is less than 100, and some twenty-five or thirty form a waiting line outside.

"A CLASS TO THE TRIAL

"The Del Rio school civics class was taken to one of the court sessions by its teacher, Miss Mary Louise Nelson. It prompted the remark that it should have been the physiology class. Another suggested taking the vocational agriculture class, referring to the goat glands.

"Judge McMillan keeps the strictest of order and there hardly has been a witness or attorney whom the judge has not called down in some manner. . . .

"Members of the jury represent varied professions, with ranching, as might be expected, predominating. They are: J. D. Harwood, Bracketville ranchman; Daniel Pedrotti, Del Rio merchant; H. D. Dick, Knippa farmer; Wilson Bonner, Dryden ranchman; William Paysinger, Batesville ranchman; L. G. Nunn, Uvalde bookkeeper; H. B. Cummings, Reagan Wells farmer; I. Hayes Cavender, Con Can stock farmer; B. H. Dessler, Juno ranchman; Herman Schafer, Brackettville, laundry employee; Harold Davis, Crystal City laborer; and Chester N. Neely, Sabinal mechanic."

The jury was chosen from some thirty residents of the Western District, most of them ranchers and farmers of this area, some representing various businesses. After preliminary addresses to the jury by the attorneys, the lawyer for the plaintiff presented as exhibits the articles published in *Hygeia* for January and February 1938.

TESTIMONY OF EFFIE KELLEY

The first witness, Miss Effie Kelley, stated that she was a newsdealer in the city of Del Rio and that the periodical had been circulated in the town.

TESTIMONY OF A. C. PETERMEYER

The second witness was A. C. Petermeyer, who stated that since April 15, 1938, he had resided in Little Rock, Ark., and before that time, from October 1933 to April 1938, in Del Rio, Texas. He stated that he was a physician and surgeon, licensed in May 1917 in Missouri; by reciprocity in 1918 in Kansas, where he became associated with the Brinkley Hospital in Milford in October 1933. He moved with the Brinkley group to Del Rio, where he remained until removal to Little Rock in 1938. He obtained his license in Texas in 1929. After stating that he had observed the work of Dr. Brinkley during his association, the evidence proceeded as follows:

Q.—Now, tell us, Dr. Petermeyer, please sir, what specialty, if any, Dr. Brinkley engaged in while here in Del Rio? *A.*—His specialty was dealing more with the infections and hypertrophy of the prostate gland, in conjunction, of course, with the treatment and surgery of other urinological conditions. By that I mean urinological—I mean the urinary organs, the kidneys, the bladder and urethra.

Q.—And was Dr. Brinkley or not during all of that time engaged in the active practice as a physician and surgeon? *A.*—He was.

Q.—Was he or not during that time himself performing operations on patients? *A.*—The majority of them.

Q.—For prostatic trouble? *A.*—Yes.

Q.—And cases of that nature? *A.*—Dr. Brinkley was performing practically all of the operations.

Q.—Now, tell us, please, Doctor, what hospitals he has in Little Rock, Arkansas, please, sir? *A.*—Dr. Brinkley has a hospital at Twentieth and Schiller Avenue in the city of Little Rock, and a second hospital on the Arch Street Pike, about sixteen miles from the city of Little Rock.

Q.—Tell us what the nature of the practice in those hospitals is and what they are operated for, particularly for what purpose? *A.*—In the two hospitals there are these departments, the urinological department, which treats, examines and treats and operates conditions of the urethra, urinary tract, kidneys, bladder, prostate gland, testicles; and the rectal department, where all pathological or disease conditions of the rectum and colon are treated. We have a hernia department where we specialize in the injection treatment of hernia. Those are the three departments that we have.

Q.—Now, how many hospitals—I mean is that all in one hospital? *A.*—The majority of that work is done at the hospital at Nineteenth and—or Twentieth and Schiller. The hospital in the country is used more for our patients who have had work to come back for free examinations and check-ups and to receive additional treatment should any of them be in need of it, but the hospital on Schiller sometimes is filled to capacity so that new patients arriving at that hospital are sometimes taken care of at the hospital in the country.

Q.—Tell us what personnel there is at the hospital there, Doctor, with reference to doctors and help in the way of nurses. *A.*—At the city hospital Dr. Brinkley and Dr. Osburn are in charge of all the urinological treatments and surgery. Dr. H. J. Hilburn specializes in diseases of the rectum; also the treatment for varicose veins and hernia. Dr. Davis is in charge of the physical examinations, Dr. J. H. Davis makes all of the physical examinations of the new patients coming to the town hospital. Dr. Chandler is in charge of the x-ray department and chemical laboratory. Mr. Kirschberg is the laboratory technician. There are two or three more whose names I am not familiar with who are working in the laboratory under Mr. Kirschberg. Mr. Kirschberg is a licensed laboratory technician. At the country hospital Dr. Parnell is in charge of the patients who arrive there for their rechecks and treatment. Dr. Hilburn reports there every afternoon to treat rectal and hernia cases. When the city hospital is filled to capacity and new patients are then taken to the country hospital where they are operated by Dr. Osburn. Miss Vira Wederbrook is superintendent of the town hospital. She is the surgical nurse and assistant to Dr. Brinkley and Dr. Osburn. We have a staff of graduate nurses, the names of whom I am not able to give you.

Q.—Yes, sir. *A.*—But there are twelve or more graduate nurses.

Q.—Now, Dr. Petermeyer, can you tell us who of those doctors were with Dr. Brinkley here in the hospital here? *A.*—Dr. J. H. Davis, who is examining physician at the town hospital in Little Rock, was also examining physician at the hospital in Del Dio; Dr. Chandler, who is in charge of the x-ray department in Little Rock was also in charge of the x-ray department while the hospital was located in Del Rio; Mr. Kirschberg, a licensed technician, was also in Del Rio.

Q.—Now, Doctor, can you tell us, please, sir, what the extent or volume of the practice of Dr. Brinkley was here in Del Rio? *A.*—You mean the number of patients?

Q.—Yes, sir. Just give us an idea of the volume of his—and extent of his practice and where they came from while he had his hospital here? *A.*—While the hospital was located in Del Rio, Texas, patients came to the Brinkley Hospital from every state, practically every state, practically every province in Canada, Alaska, Nova Scotia and Central America.

Q.—Well, were the— *A.*—The number of patients during that time from October 15, 1933, to January 1, 1938, would be somewhere in the neighborhood of fifteen thousand.

Q.—Now, for what length of time was that fifteen thousand, over what period? *A.*—From 1933, about October, to January 1, 1938.

Q.—Now, do you know whether or not you had many patients from this general locality in and around Del Rio? *A.*—Yes, we had more patients from the state of Texas than any other locality.

Q.—A great many more from Texas than other places? *A.*—Many more.

Q.—And a great—did you or not have a great many patients from the general region around Del Rio, in and around Del Rio? *A.*—Yes, we had a large number of patients from Del Rio and Val Verde County and surrounding counties.

Q.—Now, Dr. Brinkley has a license to practice in Texas *A.*—Yes.

Mr. Brown:—I don't think this gentleman would know about that.

THE COURT:—The license would be the best evidence.

Mr. Morris Jr.:—You admitted that in your answer. Do you make any question about his having his license to practice?

Mr. Brown:—We just have the formal objection, if the court please, that this gentlemen wouldn't be able to testify to that.

THE COURT:—If there is no issue about it why waste time on it? Is it admitted he is licensed to practice in Texas? We have plenty of issues in the case without wasting time. Is there any admission on that?

Mr. Brown:—No. I just didn't want this gentleman to get out of the general field.

THE COURT:—I think we have lost time by the objection. Go ahead.

Q.—And do you or not know that during all that time he was engaged in the active practice of his profession? *A.*—He was.

Q.—And is he yet engaged in the practice as a physician and surgeon? *A.*—He is.

Q.—Did you know anything about the article which was published in *Hygeia*, the article herein sued on, at the time it came out, about that time? *A.*—Yes, I read it.

Q.—And did you or not have any occasion to discuss the matter or observed Dr. Brinkley with reference to the article? *A.*—Yes.

Q.—And what affect it had on him? *A.*—Yes, sir.

Q.—Just tell the jury, please, sir. *A.*—Well, the—

Mr. Brown:—Your honor, I don't remember if there is any issue as to what effect it might have had on him personally.

THE COURT:—I think he claims he suffered mental anguish.

Mr. Brown:—All right.

Q.—Go ahead, tell what you observed in that connection, just tell the jury. *A.*—There was a great deal of conversation on the street by the people of Del Rio. A large number of copies were requested from our news stands so that people could read it. The article humiliated Dr. Brinkley in the presence of his friends and acquaintances in Del Rio, as well as with the members of his profession.

Q.—Did it or not appear then to bother and distress him a great deal? *A.*—It did; Dr. Brinkley was rather reluctant to appear in public after that article was published about him. Naturally it would make any one feel reluctant to appear in public after that.

CROSS EXAMINATION

Questions by Mr. Brown:

Q.—Dr. Petermeyer, what college is it, please, that you graduated from? *A.*—The American School of Osteopathy and Surgery, at Kirksville, Mo.

Q.—Did they specialize there principally in bone work? *A.*—No; the curriculum at Kirksville is practically the same as any class A school; the subjects taught and textbooks used are standard ones.

Q.—But it is known as a college of osteopathy, is it not? *A.*—It is.

Q.—Did you perform the surgery after you came to Del Rio, in Dr. Brinkley's hospital? *A.*—I was doing the surgery, part of it, when I came, but the majority of it was done during 1936 and 1937.

Q.—That is after you had been here some time? *A.*—Yes.

Q.—Did you ever do that operation where they take a part of a goat and put it in a man? *A.*—No.

Q.—Do you operate in the hospital up in Arkansas? *A.*—No.

Q.—What is the principal type of operation that they do when a man has a complaint, as far as his prostate glands are concerned? How do you operate for that ordinarily? *A.*—Do you want a complete description of the operation, how it is performed?

Q.—Yes, if you please. *A.*—After the patient has been accepted for treatment and is in need of treatment to the prostate gland, and all of the preoperative treatment has been performed, and the patient has been prepared properly to receive an operation—I will just give you the technic of the surgeon in the operating room. That is what you want?

Q.—In words we can understand, if you please. *A.*—The scrotum is anesthetized with apothesine.

Q.—With what? *A.*—Apothesine, a local anesthetic. The testicle is picked up in the left hand with the index finger of the right hand; the head of the epididymis is an appendage of the testicle; and an incision is made through the skin, through the dartos, through the fascia, through the cremasteric muscle—

Mr. Morriss:—Tell us what those are.

Q.—That is just cutting down to get to the testicle? *A.*—Yes. An incision is made through the scrotum down to the testicle. The testicle is lifted up into the incision so that the epididymis is available.

Q.—Tell us just in language that we may understand better. What is the epididymis? *A.*—It is a collection, you might say, of tubules attached to the side of the testicle. It has two parts.

Q.—Kind of like an appendage? *A.*—You might consider it something like that. It consists of a large area known as the globus major and a tail of it known as the globus minor. From the globus minor we have an extenuation of a tube known as the vas deferens.

Q.—That runs still on up? *A.*—That runs on up to the seminal vesicles. After the testicle is brought up into the incision so that the—this appendage, the epididymis, can be thoroughly examined, the ducts which lead from the testicle to make up the epididymis are inspected and examined with the examining finger.

Q.—Now, excuse me. I am not very familiar with it, of course. Is there a little space, a little distance between the main bulk of the testicle and where the addition or appendage is? Is there a little space there? *A.*—Yes, sir, slightly.

Q.—About how much is that, ordinarily, just in a general way, those little tubes, about how long would they be ordinarily? *A.*—Which tubes are you speaking of?

Q.—The ones reaching from the testicle, that you mentioned, up to the epididymis? *A.*—They are very short, perhaps 1 centimeter in length after they leave the testicle to where they converge in the epididymis. In fact, the epididymis is so closely attached to the testicle, it is closely attached to the testicle with tissue, but you can feel with your finger the space between the epididymis and the testicle.

Q.—Now, what do you do then in respect—I interrupted you there. What do you do with respect to examining those little tubes? *A.*—We examine them with the fingers to feel for the presence of small nodular areas, which usually mean infection.

Q.—What do you call those little tubes, technically? *A.*—The vasa efferentia.

Q.—Now, is there another word they use for it, some kind of efferent tubes? *A.*—It is the same thing.

Q.—Efferent tubes? *A.*—Yes.

Q.—Now, how many of those, ordinarily, in each testicle? *A.*—Fifteen to twenty.

Q.—Well, after you have then examined and found which one is diseased, if any, and the condition, what do you do then in continuation of the operation? *A.*—They are ligated.

Q.—What does that mean, please? *A.*—Tied off.

Q.—Well, is the number in each testicle in every person always the same? *A.*—They may vary.

Q.—Do they vary in different people in number, I mean? *A.*—Yes.

Q.—Well, how many do you tie off ordinarily? *A.*—Sometimes all of them and sometimes half of them and sometimes none.

Q.—I say ordinarily, an ordinary operation? *A.*—The ordinary operation, seven or eight of them are ligated or tied off.

Q.—Substantially half of them? *A.*—Yes.

Q.—Does that tied off mean—does that mean that no fluid or anything can go through that one any more? *A.*—Yes, sir.

Q.—It stops it? *A.*—Yes, sir.

Q.—Well, if you stop all of them in each testicle, what result would that have? *A.*—Well, on what?

Q.—I mean on the man's sexual powers. For instance, what would be the effect of it? *A.*—Sometimes it increases it.

Q.—To tie them all off? *A.*—Yes, sir, all of them.

Q.—All of the tubes? *A.*—Yes, sir.

Q.—I mean, now, as to sterility? *A.*—It would produce sterility, but at the same time increase his sexual vitality.

Q.—What is the difference between sterility and impotency? *A.*—Sterility means that the individual is not able to produce a heavy male sperm, which when it comes in contact with the female ovum will cause a direct cell formation.

Q.—In other words, he can't have any more children? *A.*—That is right.

Q.—But that condition can exist and at the same time he would have the ordinary sexual power except for the production of children? *A.*—He could have.

Q.—But if you cut them all off on both testicles that would produce sterility? *A.*—It would.

Q.—And in the ordinary operation you cut off about half of them? *A.*—Yes.

Q.—Of course, that varies in the different patients, I imagine? *A.*—Yes.

Q.—Now, what else do you do now in addition to cutting off some or all of these little tubes that run between the main part of the testicle and the appendage there? *A.*—The artery running along the vas deferens is also ligated, tied off.

Q.—That is the blood vessel that runs along the tube that goes on up from the testicle, that goes on up? *A.*—Yes, sir. The vas deferens is picked up and injected with about 5, 3 to 5 cubic centimeters of a 2 per cent mercurochrome solution. This solution is injected through the vas deferens and if the tube is open and functioning the solution will pass on into the seminal vesicles.

Q.—What is that last you mentioned? *A.*—Seminal vesicles?

Q.—Yes. *A.*—Small vesicles are located between the rectum and bladder, just above the prostate, and is a sort of reservoir or storehouse for seminal fluid. As it passes upward from the testicles this solution reaches the seminal vesicles, if they are distended with it considerably, and out through the ejaculatory ducts. These two little tubes leading from the seminal vesicles lead to the prostate. In fact, they go through the posterior aspect of the prostate gland, emerging on the floor of the prostate. The solution usually appears the next time the patient voids. At least a part of it does. It does away with the catheterization of the ejaculatory ducts, which are often blocked with infection. If the solution will not pass into the seminal vesicles or if we find the vas deferens is blocked and it presents plenty of evidence of infection we may remove a portion of it.

Q.—Of what is that? *A.*—Of the vas deferens.

Q.—Why? *A.*—Because it is like any other focal infection, like the appendix or tonsils or any other organ, if it can no longer function it becomes a menace to the future health of the individual.

Q.—And you cut out a portion of them? *A.*—Yes, sir.

Q.—About how long in length is that, ordinarily? *A.*—It may be from an inch to 4 or 5 inches in length.

Q.—Does that also produce sterility. *A.*—A patient is already sterile, if the mercurochrome will not pass through the patient is sterile.

Q.—Is that all of the ordinary and usual operation that you do for the condition? Now, what effect—excuse me—what effect does that operation have on an enlarged or diseased prostate gland? *A.*—Research workers and scientists who have been working along the lines of the glandular system have fairly well agreed on the fact that there are two secretions from the testicles. The one secretion, or excretion—

Q.—Excuse me, please. I meant to say that as far as the enlarged prostate is concerned, the operation you mentioned there, whether that will eventually cause the prostate to become smaller, be relieved as far as the passing of urine is concerned? *A.*—That is what I was coming to. I was only explaining it a little more in detail.

Q.—Yes, sir. Now, then, in reference to—

Mr. Morriss Jr.:—Let him answer the question.

Mr. Brown:—You can ask him.

Q.—If the prostate is enlarged so that it blocks the passage of the urethra do you ever cut a portion of it out? *A.*—If the prostate is enlarged enough to produce over 4 ounces of residual urine—by that I mean the gland is enlarged so much that it will not permit the bladder to empty.

Q.—Then what do you do? *A.*—A transurethral resection is necessary.

Q.—What is that? *A.*—That is the removing of the obstructing portion of the prostate.

Q.—Do you have to make an incision? *A.*—No; the instrument is passed through the urethra and the operation is performed through the sheath of the instrument you use.

Q.—Now, does Dr. Brinkley's hospital ordinarily charge for—what does it ordinarily charge for that operation? *A.*—Transurethral resection? We usually charge $250.

Q.—And for this operation you described first, about the testicle? *A.*—From $275 up.

Q.—Does he sometimes give a guaranty after one of these operations? *A.*—We give future service, yes, sir, and we guarantee to the patient that if the operations that he has received does not reduce the growth of the prostate gland so he can empty his bladder properly he is privileged to return and have a transurethral resection at no additional expense.

Q.—Does that cost $750? *A.*—Yes, sir. That also includes his hospitalization and his medications and dressings, anesthetic and operating room fee.

Q.—You mentioned a while ago a class A medical school. What do you mean by that, please? *A.*—The medical schools are classified, I think, A and B, and an A school having a higher rating than a B school.

Q.—Do you know who it is that classifies the different medical schools throughout the country? *A.*—No.

Q.—Don't you know, Doctor, that is—that it is a fact that they are classified in that classification as accepted by the American Medical Association? *A.*—I presume so.

Q.—That is your understanding of it, is it not? *A.*—Yes.

Q.—Now, up there at the hospital there, as I understand it you are now no longer serving as a doctor, but you are the business manager, are you? *A.*—Yes.

Q.—Do you know of any other recognized doctors in the United States that use the technic of operation that you have here described? *A.*—Yes, sir.

Q.—Who are they, please? *A.*—I do not have the consent to divulge their names, but I can give you one whose article has appeared in literature published by the institution.

Q.—By which institution do you mean? *A.*—From the Crile Clinic in Cleveland, Ohio.

Q.—That is the institution that published what, what was it? *A.*—The institution published this article, the article was written by Dr. Lower, who is in charge of the George Crile Clinic.

Q.—At what place? *A.*—Cleveland, Ohio.

Q.—You don't want to name any others that do this same operation that you all do? *A.*—Not having their permission to divulge their names, I wouldn't care to.

Q.—All right, sir. Now, as I understood it, you said you operated on about 15,000 people here at Del Rio? *A.*—No, that number of patients came to our hospital.

Q.—Oh, I see. Well, of that number, how many were operated on, do you remember? *A.*—No, I do not.

Q.—Do you know what the average charge for the operations was while they were here in Del Rio? *A.*—The average charge would be including their hospitalization and operating room fee and anesthetic and examination. Their entire expense would probably average around $400.

Q.—Did Dr. Brinkley or any one in his hospital, after they got here to Del Rio, did they ever use the goat gland? *A.*—No.

Q.—Transplanting the goat gland in a human? *A.*—No.

Q.—By the time they got here they had quit that, had they? *A.*—There was none of that done here.

Q.—Are you doing any of that up in Arkansas? *A.*—No.

Q.—Did you ever do any of that yourself in Kansas when you were there at Milford with the doctor? *A.*—No.

Q.—Did you ever see any of it done while you were there? *A.*—No.

Q.—The man that wrote that article up there for the Crile Clinic saying he had done this same operation, did you mean cutting out a portion of the prostate gland or the operation you first described, that is, where you tied away the little tubes? *A.*—A ligature operation, yes, sir.

Q.—That is the one he described in that article, is it? *A.*—Yes, sir.

Q.—Do you know whether or not that doctor also put the mercurochrome in the tube also, like you do? *A.*—I don't recall.

Q.—Do you get more patients with this trouble of the prostate from the cold states or warm states, or is there any difference? *A.*—There is a difference, because cold weather affects urinary troubles more than warm weather, and we have a great many patients, of course, from both regions, warm and cold, but cold weather does affect and aggravate the symptoms of the prostate and bladder.

Q.—Also do you get more people from the north of you or south of you, from Little Rock? *A.*—I don't believe there is very much difference.

Q.—Do you know about how many patients they operate on there now in Little Rock since you have been there, how many they operate per week, for this ordinary operation? *A.*—During the past two months I expect the average would be about fifty, between forty and fifty a week.

REDIRECT EXAMINATION

Questions by Mr. Morriss Jr.:

Q.—You spoke of the operation when all of the ducts are ligated as producing sterility. Do you or not ever perform any operation which might produce or continue a sterile condition in a man without first fully explaining that situation to him and obtaining his consent? *A.*—No, we do not.

Q.—Is that the universal practice in that respect? *A.*—It is.

Q.—Well, now, is that usual in the case of very old men? *A.*—Altogether.

Q.—Now, Doctor, you also told us that a charge of $750 was made which covers the operation and hospitalization. Does it or not also include the examination which is made? *A.*—Yes, it does.

The witness then outlined what was said to be the regular physical examination of patients coming to the Brinkley Hospital, beginning with the top of the head and through the body, as is usual in complete physical examinations, describing also the basal metabolic test, the use of the cystoscope, electrocardiograph, and the x-rays. He also described in detail the equipment of the institution, proceeding then as follows:

Q.—Now, then, Dr. Petermeyer, I want to ask you this question, please, sir: Counsel asked you whether any other type of operation was performed and you spoke of the transurethral resection. *A.*—Yes, sir.

Q.—Will you explain to the jury what that consists of and how that operation is done?

THE COURT:—I think he explained that on cross examination.

Mr. Morriss Jr.:—I didn't understand it so.

THE COURT:—Counsel asked him what it was and he told him what the operation was. Now, you have had this witness on the stand for a long time. If you are going to back-track and repeat we will never get through.

Mr. Morriss Jr.:—I wasn't back-tracking—

THE COURT:—I am not arguing with you, Counsel. That is the ruling of the Court.

Mr. Morriss Jr.:—Yes, sir. Note the exception.

Q.—How many different types of operations would you say are done with reference to the prostate, then? *A.*—Aside from our routine prostate operation we do that and also the suprapubic operations. That is opening into the bladder above the pubic bone, openings there are made to remove large stones and growths. We also remove stones by a crushing instru-

ment which is introduced through the urethra, through the urinary canal, providing the stone is not too large. The instrument will remove a stone about 3½ centimeters; that would be about, well, say about an inch or an inch and a half in diameter. The instrument will pick up a stone that size and crush it, and by crushing each fragment separately it can be washed out through a tube or sheath.

Q.—Are there any other operations that are performed, Doctor? *A.*—Operations on the testicle, scrotum, penis, kidney, bladder.

Q.—Now, in my questions of you I have intended these questions to apply to the time that this article was published, and let me ask you whether or not your answers to these questions, whether the same answer is applicable to the time which the article was published, as to the various operations and examinations given and treatment? *A.*—It is.

Q.—And the technic pursued? *A.*—The same.

Q.—Now, how long, Dr. Petermeyer, have you been observing Dr. Brinkley in the performance of prostate operations? *A.*—Since 1933.

Q.—And have you or not assisted him during that time with operations? *A.*—Prior to the time he located his hospital in Little Rock?

Q.—Yes, sir, that is what I say, while he was here in Del Rio? *A.*—Yes, sir.

Q.—You assisted him with the operations, did you, Doctor? *A.*—Yes, sir.

Q.—And did you or not in many other instances in which you did not assist, observe the operations? *A.*—Yes, sir.

Q.—And can you tell us how many operations you have seen Dr. Brinkley perform for prostate trouble? Just give us your best estimate. *A.*—Oh, several thousand.

Q.—Now, Doctor, have any of these patients who have come to Dr. Brinkley originally and received any of these operations, have they returned during the time you have been with the hospital? *A.*—They have.

Q.—And have you or not had an occasion to examine and observe them prior to their first operation by Dr. Brinkley? *A.*—Yes, sir.

Q.—And have you or not had occasion to observe and examine them subsequent to the operation? *A.*—Yes, sir.

Q.—Well, now, just tell the jury as best you can what the practice with reference to the patients is with reference to returning, and approximately how many you would say that you have had the occasion to observe, and how long after they had been to Dr. Brinkley was it that they would come back? *A.*—From 1933, until January of 1938, I had occasion to examine a large number of patients who came to the hospital, to give them the preoperative treatment and either see them operated by Dr. Brinkley or operate them myself, give them the postoperative treatment and check them out or dismiss them from the hospital at the end of their hospitalization time.

THE COURT:—That wasn't the question he asked you, Doctor. He asked you about those that came back.

A.—Oh, yes. Our patients are privileged to return for rechecks, for examination. We want them to, and if they don't return in a certain length of time we frequently write them and ask them why they haven't come back for their examination or check-up. I would say that 5 per cent, about 5 per cent of the patients return for additional work.

Q.—Now, then, after what length of time have they been out, ordinarily, just tell us what varying lengths of time. *A.*—The time varies from six months to several years.

Q.—Now, then, Doctor, tell us with reference to those patients which you have had an opportunity to examine and observe prior to the time of their operation and subsequent to the time of their operation, what you have found with reference to beneficial effects of the operation given? *A.*—I would say that 80 to 95 per cent of the patients respond favorably to the treatment.

Q.—Well, now, tell us what you mean by responding favorably, what effect it has, what physical effect? *A.*—Those who have received our routine operation and treatment for infection or enlargement of the prostate gland?

Q.—Well, I am talking about the gland, what effect you have observed in the way of improvement or benefits received? *A.*—The benefit has been general. They have been not only able to empty their bladders better and be relieved of their urinary symptoms, but they have felt better and in a general way have gained weight and their rest is improved and they sleep better. Many of those who were not able to work at all are able to go home and within a reasonable length of time go back to their job.

Q.—Do you mean by that, then, Doctor, that the operation was successful in those percentages of cases that you have told us? *A.*—Very.

The witness then described the method by which records are kept in the hospital, after which the evidence proceeded.

Q.—Now, Doctor, please explain to us—counsel asked you about it, about the guaranty. Just what that consists of, what it entitled the patient to, explain that to us? *A.*—The guaranty, or lifetime future service, entitles the patient to return at any time regardless of time for free examination and check-up. Should we find that he is in need of further treatment we offer it to him at no additional expense. If he needs a transurethral resection, that is performed without any additional expense other than his board and room in the hospital, but he never pays any more for treatment or surgery pertaining to the type of work that he came for.

Q.—Then in these cases, Doctor, is it not true that when they first come you make the complete examination and then the operation or treatment is given and then is the patient also requested to return within six months or a year for an additional check-up and to return regularly? *A.*—No, he is not. It is not compulsory, he does not have to return, but we ask him to and request that he does.

Q.—Yes, sir, that is what I say, that he is requested to return for additional examination and check-up to see how his general physical condition is getting along, is that correct? *A.*—Yes, sir.

Q.—And that additional examination and check-up and any further treatment which may develop to be necessary of the prostate is all administered without additional charge, is that right? *A.*—That is right.

Q.—Doctor, about how long do the patients remain in the hospital after the operation? *A.*—In uncomplicated cases they usually remain about seven days.

Q.—And how long are they there before the operation? *A.*—That depends somewhat on the time necessary to make the examination and depends on the condition of the patient, whether or not he is an operative risk at that time. Occasionally we have to keep a patient several weeks, or into the second week or several weeks, before he is operated, and occasionally at that length of time we send him home without any service because we feel he would be a poor operative risk.

Q.—You mean his physical condition is determined by you as such that you feel you could not help him or benefit him with an operation? *A.*—We feel we could not help him and he might die as a result of the operation from shock or from being in a debilitated condition.

Q.—Because of his debilitated condition? *A.*—That is right.

Q.—In those cases do you make a charge to the patient? *A.*—We make a charge of $50 for the examination plus $35 a week in the hospital.

Q.—Is that all of the charge that is made to them? *A.*—We charge sometimes for medications and sometimes we charge just for the examination and their hospitalization.

Q.—Now, with reference to the injections of the mercurochrome solution, Doctor, in the vessels, as you have described it, just tell the jury where that is injected and what is its course?

THE COURT:—No, he has described that, counsel, once.

Mr. Morriss Jr.:—I want to know the purpose of it.

THE COURT:—If you are going to go over the testimony of every witness like you have on this doctor we would be here too long. I want you to bring this examination to a close. You have brought him way beyond the realm of redirect examination.

Mr. Morriss Jr.:—Well, it is—

THE COURT:—You will have to comply with the Court's rule.

Mr. Morriss Jr.:—I wasn't going to ask that question, I was going to ask in connection with what was the purpose.

THE COURT:—He has told why he did it.

Mr. Morriss Jr.:—Has he explained that?

THE COURT:—Yes, sir.

Mr. Morriss Jr.:—Note the exception.

THE COURT:—Exactly why they did it, what they did and why they did it. It is going over the same thing twice.

Q.—Is the system of examination and treatment of the patient after you have—as you have given it to us, Doctor, is that the system which was inaugurated and required by Dr. Brinkley in the treatment of prostatic troubles? *A.*—Yes, sir.

RE-CROSS EXAMINATION

Questions by Mr. Brown:

Q.—Doctor, at any hospital they give a patient a complete examination before they operate on him, don't they, and they take his history? *A.*—I think they do.

Q.—And they make a chart of all the patient tells them and of the conditions they have found, don't they? *A.*—They do.

Q.—Do you know what this formula 1020 has in it? That is Dr. Brinkley's formula, isn't it? *A.*—Yes, sir.

Q.—Do you know what he has in it? *A.*—Yes, sir.

Q.—What is that? *A.*—I haven't his permission to answer that.

Mr. Brown:—We would like to know.

THE COURT:—You will have to answer the questions, Mr. Witness.

Q.—How much do you charge for that No. 1020?

THE COURT:—Wait a minute, they have some objection.

Q.—How much charge is made for that prescription 1020? *A.*—Of course, a treatment with that intravenous medication we charge $100.

Q.—Is that the one you have given the patient to take home and have it put in him by his home doctor when he gets home? *A.*—No.

Q.—Postoperative treatment? *A.*—No, we give that to him at the hospital.

Q.—The doctor at home doesn't use 1020? *A.*—Only if he is a recheck patient and does not have time to remain longer.

Q.—But anyway you charge $100 for prescription 1020? *A.*—Not for that if he takes it home, if he takes it home there is no charge.

Q.—If it is given in the hospital there is a charge of $100? *A.*—Yes, sir.

Q.—What has it got in it?

Mr. Morriss Sr.:—Of course, this witness says he is an employee of that hospital and this is a proprietary—

THE COURT:—I know, but if he knows it. He is here testifying on the stand. He will have to answer the question.

Mr. Morriss Sr.:—It is privileged.

THE COURT:—There is nothing privileged about it.

Mr. Morriss Sr.:—Would it not be privileged in a sense in that it is a matter that belongs to his employer and—

THE COURT:—No, I don't think so.

Mr. Morriss Sr.:—In other words, a trade secret.

THE COURT:—I don't see any secret. He said he gives it to the patient to take home, and chemists could analyze it.

Mr. Morriss Sr.:—Note the exception.

THE COURT:—Answer the question if you know.

A.—The chief element in it is hydrochloric acid.

Q.—What per cent of it is hydrochloric acid? *A.*—I don't know.

Q.—Very small, is it not? *A.*—No, sir.

Q.—Do you have any idea of the percentage? *A.*—Perhaps one to one thousand.

Q.—One to one thousand. In other words, one thousand of what, water? *A.*—Yes.

Q.—One thousand of water and one of hydrochloric acid? *A.*—Yes.

Q.—Is that all it has in it? *A.*—No.

Q.—What else has it got in it? *A.*—Mercury.

Q.—How much mercury? *A.*—I don't know the percentage.

Q.—What else has it in it? *A.*—It has methylene.

THE COURT:—Are you talking about now or at the time?

Mr. Brown:—At the time, if you please.

A.—Methylene.

Mr. Brown:—At the time or before?

THE COURT:—You had better make that clear because the evidence shows Dr. Brinkley is running a place up in Arkansas, and we are only interested in what happened down here.

Mr. Brown:—This article was in February of last year, and I am talking about either at that time or a reasonable time before that.

THE COURT:—All right.

Q.—What else has it in it, please? What else had it in it, please? *A.*—I believe that is about all.

Q.—How much did you have to pay, wholesale, for that stuff you put in 1020? *A.*—I don't know.

Q.—Now, do you know what was in these other prescriptions that he prescribed over the radio, prescriptions No. 50 and 60 and those other prescriptions he would talk about over the radio and the person would go to the drug store and get the prescriptions by number so and so? Do you know what is in those prescriptions? *A.*—No, sir.

Mr. Morriss Jr.:—We want to object to that question as assuming something not proved. It is a duplicitous question also.

THE COURT:—Yes, it is not in line with anything he asked on redirect examination. I don't think it is proper re-cross examination of any matter.

Mr. Brown:—Yes, sir.

Q.—Do you know what gives that 1020 the blue color to it? *A.*—The methylene, I believe.

Q.—Now, what pathologist is it there at your hospital, or was it at the time of this article and before, what pathologist was it that made this investigation of the tissues and so forth? *A.*—Mr. Kirshberger.

Q.—Mr. Kirshberger? *A.*—Yes.

Q.—He is a doctor? *A.*—A licensed technician.

Q.—Who licensed him, do you know? *A.*—Oh, no.

Q.—Do you know of anybody—is there such a thing as a licensed laboratory technician? *A.*—Well, these technicians who graduate from some of these standard laboratories.

Q.—You don't know who licenses them? Do they get licenses from the state, or whom? *A.*—I don't know.

Q.—Now, is this Dr. Brinkley's hospital, or was it at the time here and before, was his hospital approved by the American Hospital Association?

Mr. Morriss Jr.:—We object to that as being irrelevant and immaterial and having no bearing upon the question.

THE COURT:—Overrule the objection.

Mr. Morriss Jr.:—And as being hearsay in its nature, and without any probative force and prejudicial to the rights of the plaintiff.

THE COURT:—If the witness knows I think it is legitimate examination.

A.—I don't know.

Q.—Was it approved by the American College of Surgeons?

Mr. Morriss Jr.:—The same objection.

THE COURT:—What is the witness's answer?

A.—I don't know.

THE COURT:—He says he doesn't know.

Q.—As I understand it, Doctor, you operated while the hospital was here in Texas, did you? *A.*—Yes, sir.

Q.—And was Dr. Osburn here at the hospital at that time? *A.*—No, sir.

Q.—Did he ever work here at the hospital, Dr. Osburn? *A.*—In the business office.

Q.—But he didn't operate, did he? *A.*—No.

Q.—But he was the business manager in Texas and you operated, is that right? *A.*—Yes, sir.

Q.—Then when you moved to Arkansas you changed it there, he operates there in Arkansas and you are the business manager, is that right? *A.*—Dr. Brinkley and Dr. Osburn.

Mr. Morriss Jr.:—We object to that as being immaterial and irrelevant, anything that may be done after the article in question was published.

THE COURT:—I don't know, it goes to the qualifications of these parties.

A.—Dr. Brinkley—

THE COURT:—Just a moment. It goes to the qualifications of these parties, to develop their qualifications. Overrule the objection.

Mr. Morriss Jr.:—Note the exception.

A.—Dr. Brinkley and Dr. Osburn.

Q.—Now operate in Arkansas? *A.*—In Little Rock.

Q.—And you are the business manager? *A.*—Yes, sir.

Q.—Do you make the arrangements to get the money out of the folks before you do the work on them? *A.*—No, sir.

Q.—You do it for nothing, without getting any money? *A.*—The patient pays the business manager of the hospital. I am at the business office and have nothing to do with the collection of fees.

EXHIBITS

The attorney for the plaintiff then presented in evidence as exhibit the issue of *Hygeia* for January 1938 containing the first portion of the article entitled "Modern Medical Charlatans," and particularly the portion which read: "In a few spots over the United States, around 1925 to 1930, local stations continued to pour forth filth and falsehood. In the obscure Kansas village of Milford, a blatant quack, one John R. Brinkley, whose professional record reeks with charlatanism of the crudest type, had for some years been demonstrating the commercial possibilities of goat-gland grafting for alleged sexual rejuvenation."

TESTIMONY OF LOWELL BROWN

The next testimony was that of Lowell Brown. He testified that he was tutor for Dr. Brinkley's son and that he made a trip with them to Europe in 1937. He described the conduct of Dr. and Mrs. Brinkley on the boat, stating that they associated freely with the other passengers. He stated also that Dr. Fishbein was a passenger on the same boat. The evidence then proceeded:

Q.—Well, did any of your party associate with the people on the boat, with his people or any of them? *A.*—Yes, I think Dr. Brinkley's son and Dr. Fishbein's boy played together for two or three days until they knew whose sons they were. In fact, I visited with Dr. Fishbein's daughter and played ping-pong with her a few times, and we drove a golf ball into a blanket hanging on the ship there, and along about the second or third day I visited with her I casually remarked to her if she knew who my boss was and she said no, and I told her and she was interested in asking what kind of man Dr. Brinkley was.

THE COURT:—Counsel, is this competent.

Mr. Morriss Sr.:—I don't know where he is leading, your honor. I don't know whether it is or not, but it may be.

THE COURT:—I know, but it seems to be some narration of an episode between him and a girl.

The Witness:—Well, it is—

THE COURT:—Just a moment, Mr. Witness, you be quiet until the Court has colloquy with counsel and confine yourself to answering questions. What do you say about it, counsel?

Mr. Morriss Sr.:—I am sure that it would be of no moment unless something came of it. I will ask him this:

Q.—Did that association continue on the trip throughout, or was it terminated, or what? *A.*—She terminated the conversation rather abruptly. I think we spoke a time or two after that but never associated any more after that.

Q.—Was it or not terminated by you, on your account, or was it terminated because of her?

THE COURT:—Counsel, I don't think that is binding on Dr. Fishbein and I don't think it is relevant unless you can show something with regard to the article itself.

Mr. Morriss Sr.:—This was before the article, your honor, this is some time before.

THE COURT:—I know that, but the charge in the article was on the ship they didn't associate with other people. Now you are trying to prove what this tutor here did.

Q.—Now, then, what about Doctor's—was the party composed of the doctor and his wife and son and you? *A.*—Yes, that is correct.

Q.—You speak of yourself, were you with his son? *A.*—Yes, sir.

Q.—His little boy? *A.*—Yes.

Q.—How old is the doctor's boy? *A.*—Eleven years.

Q.—Well, now, what about Dr. and Mrs. Brinkley on that trip back, did they or not refuse to associate with anybody, or anything of that kind? *A.*—Just the usual thing, just as I tell you. I could have told you with the other questioning. There was a picture taken, a group picture at the dinner table one evening on the *Queen Mary* going over. I don't believe any picture was taken coming back. I don't know of anything extraordinary about any of our behaviors in any way.

Q.—What do you mean, group picture? *A.*—Dr. and Mrs. Brinkley and Johnny Boy and myself, and I have the picture at home, and I am sure they do.

Q.—Were they in company with others? *A.*—Yes, sir, people all around them. Of course, the picture was taken by flashlight of us, of the family, and I think the picture will show in the background other tables besides ours. It was in the dining room.

He then stated that Dr. Brinkley was a delegate to the Rotary convention on this trip.

The cross examination follows:

CROSS EXAMINATION

Questions by Mr. Brown:

Q.—Have you ever been with him on any of his yachts? *A.*—Yes, sir.

Q.—What was the name of the yacht that you were with him on? *A.*—Well, it was called *Dr. Brinkley No. 2.*

Q.—*Dr. Brinkley No. 2?* *A.*—Yes, sir.

Q.—And where did you go on that occasion? *A.*—Well, my first yacht trip was up and down the New England coast. I got on the yacht at Port Washington, Long Island.

Q.—Did you ever see yacht No. 1, Dr. Brinkley's yacht No. 1? *A.*—Yes, sir.

Q.—Have you seen Brinkley No. 3? Is there a No. 3? *A.*—I think it is also called No. 2, but I have been aboard of it.

Q.—In other words, he has been the possessor of three fine yachts, hasn't he?

Mr. Morriss Jr.:—We object to that, it is irrelevant and immaterial and a conclusion and opinion of the witness.

THE COURT:—If you strike the word fine out the question will not be subject to objection.

Mr. Brown:—Yes, sir. What they declared on here is that he has been the possessor of three fine yachts, hasn't he, Mr. Witness?

THE COURT:—They objected to that as calling for a conclusion, whether or not he knows what a fine yacht is. Why don't you confine it to "three"?

Q.—He has owned three yachts, hasn't he? *A.*—Yes, sir.

Mr. Morriss Jr.:—We would make the further objection, your honor; for instance, they may have made some statements in the article which were true. That in no manner, or the proof of the truth of those in no manner relieves or tends to relieve the other.

THE COURT: I know, but they can prove all of their case at one time. Of course, it may be that this line of examination is objectionable, inasmuch as you didn't ask him anything about it on direct examination. However, under the new rules there is the right to recall the witness, and we can save that time here.

Mr. Morriss Sr.:—I thought it was a part not charged.

Mr. Brown:—That was charged particularly in the petition, about the yacht.

Q.—The boats that he went back and forth on to Europe were the best boats going and coming, as far as you know, weren't they fine, big ships? *A.*—He wouldn't be very apt to go in a tub, I wouldn't think.

THE COURT:—Now, Mr. Witness, I want to admonish you. You are supposed to answer questions here, not make an argument. That will be done by the attorneys.

The Witness:—I am sorry.

THE COURT:—Observe the court's ruling.

Q.—That ship, the *Normandie,* that is a big, fine ship, isn't it, Mr. Witness? *A.*—I think so.

Mr. Morriss Jr.:—We object to that as being a conclusion.

Mr. Morriss Sr.:—And it is immaterial and irrelevant.

THE COURT:—I think everybody can pretty nearly take notice of the fact that the *Normandie* is a big boat, probably a fine one.

Q.—What was the name of the other one, the one you went over in? *A.*—The *Queen Mary.*

Q.—Well, that is a big one and a fine one, too, isn't it, sir?

Mr. Morriss Jr.—The same objection.

Q.—Is that right?

The Witness:—Do I answer?

THE COURT:—Yes, go ahead and answer.

A.—I should think so.

Q.—And on the *Normandie* he had the most expensive and biggest suite of cabins, their biggest ones on the boat, didn't he?

Mr. Morriss Jr.:—Just a minute. This witness is not shown to be competent to testify to that.

THE COURT:—What is the materiality?

The Witness:—I wouldn't be—

THE COURT:—Counsel, what is the materiality in that?

Mr. Brown:—They allege that—that is part of the article upon which they allege and are suing us for damages for saying that. We just want to show that it was so.

THE COURT:—I believe the article said he travels abroad and returns to this country in the finest suites and finest boats. Overrule the objection.

Mr. Brown:—And that is the portion ——

Mr. Morriss Jr.:—Our objection is the witness is not shown to be competent to testify, and it would be a conclusion and opinion on his part.

THE COURT—About what?

Mr. Morriss Jr.:—Whether the most expensive and finest.

THE COURT:—If he doesn't know he can say so, but counsel can have him describe the suites.

Q.—How many rooms did you have in the suite, for instance on the *Normandie? A.*—We had two rooms, a room for the boy and I and a room for Dr. and Mrs. Brinkley:

Q.—Were they little bitty staterooms where you had to crawl up one over the other to get in bed, or what kind? *A.*—Nice sized rooms. You didn't climb up.

Q.—Twin beds in them? *A.*—Yes, sir.

Q.—Or little beds? *A.*—They had small single beds.

Q.—Did you have a living room and dining room in that same suite? *A.*—On one of the ships I believe we did.

Q.—Which one, the *Normandie? A.*—Yes.

Q.—And what kind of rooms did you have on the *Queen Mary? A.*—Well, we had double rooms, a room for the boy and I which had single beds and a room for Dr. and Mrs. Brinkley which had single beds.

Q.—Did you have a living room or dining room, anything like that? *A.*—No.

Q.—Those suites on those ships, Mr. Witness, were handsomely furnished, fine stuff in them. *A.*—I don't know whether I am competent to judge on that.

Q.—You know the difference between broken down furnishings and nice ones? *A.*—Things were comfortable enough both ways.

Q.—Weren't they handsomely furnished? You know enough about things that you can say whether they were furnished in fine style or not? *A.*—Well, naturally, any first class cabin on a ship of that kind is nicely furnished. In my opinion you couldn't get anything else.

Q.—Now, about how long did you work for Dr. Brinkley altogether? *A.*—Five years and two months. I believe, to be just about exact.

Q.—That is, you have been a tutor for his son for that long? *A.*—Yes, sir.

Q.—Did you start working for him after he came to Del Rio? *A.*—No, sir, I went to work for him in Milford, Kansas, just a few weeks before he came here.

Q.—Are you still working for him? *A.*—No, sir.

Q.—When did you quit? *A.*—December 1st.

Q.—Last December 1st?

The witness then stated that he is now in the life insurance business in Council Grove, Kansas.

RADIO BROADCASTS

At this time the court and the lawyers considered the question of the order of the court to Brinkley to produce either transcripts or victrola records of his broadcasts from the station in Mexico. Dr. Brinkley stated: "I don't know anything about it. I imagine the records prepared in 1938 have all been destroyed. I don't think the station keeps them over two or three months after they are filed over there."

After much discussion about getting the records or transcripts of the broadcasts, the court said:

THE COURT:—It seems to me as a practical matter that this should be handled very simply. Everybody knows Dr. Brinkley for years has been broadcasting over the radio. I hear it all the time running around in my car. I have always understood, and I presume it is true, that he doesn't personally go over and do it, that frequently he sends records over there. If that is true why not say so and if he can get the records all right. If we can't we will have to handle it some other way. Why make trouble about it?

Mr. Morriss Jr.:—We hadn't intended to make trouble about it. I wanted to explain that to the Court. We hadn't intended to make any trouble about it at all, we just didn't construe—

THE COURT:—If they are over there will you try in good faith to get them?

Mr. Morriss Jr.:—Yes, sir, we will.

Next came the discussion of the number of depositions which would be introduced by the attorneys for both sides and as to what the court would permit in the introduction of depositions.

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On the beginning of the second day, the plaintiff rested his case, at which time Mr. Matthews, attorney for the defendant, introduced a motion for a directed verdict in accordance with the new federal rules that permit the making of such motions at the close of plaintiff's evidence.

Mr. Matthews said:

However, preliminary to that, we would like to call your honor's attention to what we consider to be a contempt of this court and conduct on the part of the plaintiff here requiring that this case be discontinued. On last night the plaintiff in this case, Dr. Brinkley, over the radio that he regularly broadcasts over made derogatory statements with respect to the defendant in the case, mentioning him by name. He made derogatory statements with respect to the American Medical Association and made statements generally referring to and having a bearing upon the case now on trial. In substantiation of that we would like to call Dr. Brinkley.

THE COURT:—What is before the court about it? Is it your idea that the trial is prejudiced by what he has done?

Mr. Matthews:—Your honor, it seems to me to broadcast over a powerful radio station, one of the parties get on a powerful radio station during the trial of the case, and broadcast over it during the trial is obviously contempt of court.

THE COURT:—You make a motion and have him cited and I will hear that separately and apart from the case. If any of the jurors heard the broadcast and were swayed by it that might be grounds for a motion for a new trial.

Mr. Matthews:—What we want to do is to make a motion in contempt and call this to the attention of the court and ask that a mistrial be declared at this time.

THE COURT:—I am not going to declare a mistrial on it unless I am advised that some of the jurors heard or were influenced by it. I don't think it is wise during the course of the trial to stop and go to interrogating the jury about that.

Mr. Matthews:—I think that is correct, your honor.

THE COURT:—And the question as to whether they heard the broadcast and whether they were influenced by it is one that could come up on a motion for new trial, but if I stop now and start trying the jury out on what they heard, if anything, and whether it had any influence on them would militate against the trial. The jury ought not to be poked around during the trial of the case.

Now, with regard to the matter of whether the taking of the air and making a broadcast of that kind constitutes a contempt of court and whether civil or criminal I will hear you on that if you make a motion and require them to answer, when this case is over.

Mr. Matthews:—Now, your honor—

THE COURT:—I am not going to attempt to pass on the matter in advance, but I will certainly say to counsel for the plaintiff, who of course should have advised his client, inasmuch as you heard the court admonish the jury to refrain from talking or listening to any one about it and even not to read the newspapers; it is certainly poor judgment to go upon the air with the case, during the trial of the case. I don't suppose counsel is responsible for that.

Mr. Morriss Sr.:—First I ever heard of it is right now. Of course, we never in any manner anticipated it. We will inquire of Doctor about it.

TESTIMONY OF JOHN R. BRINKLEY

Mr. Brown, attorney for the defendant, then called to the stand John R. Brinkley, the plaintiff. The evidence proceeded as follows:

Examination by Mr. Brown:

Q.—What is your name, please? *A.*—John R. Brinkley.

Q.—Where do you live now, please, Doctor? *A.*—Del Rio, Texas.

Q.—And do you operate a hospital here in Del Rio now? *A.*—Not now.

Q.—You operate one, as I understand, in Arkansas, Little Rock, Arkansas? *A.*—Two hospitals, yes, sir.

Q.—Doctor, without stating the exact date, about how long have you been practicing medicine? *A.*—Since 1915, since 1915.

Q.—Yes, sir. Now, when did you first commence to advertise yourself as a doctor? *A.*—I think I began radio talks about our hospital in 1929.

Q.—Well, had you issued any pamphlets—first, when did you first do the goat gland operation, taking a piece of goat testicle and putting it in— *A.*—I got—

Mr. Morriss Jr.:—Just a moment. We object to that as being irrelevant and immaterial to any issue in the case, as being remote, too remote for consideration here, and as not bearing on any issue in the case.

THE COURT:—No, counsel, I think that if the plaintiff doesn't offer himself as a witness the defense under the rules has a right to call him, cross examine him just as fully as if he had taken the stand on his own account.

Mr. Morriss Sr.:—We think so, too.

THE COURT:—And that is what they have done. Now, he put the matter in issue here as to his reputation. What he has done, his qualifications, all that sort of thing, and they are entitled to search it thoroughly. So I overrule your objection.

Mr. Morriss Jr.:—I would like to be heard.

THE COURT:—I have just heard you.

Mr. Morriss Sr.:—May it please the court, there is a matter that I wish we might state to the court, remind the court of. That is, there is no evidence whatever from the plaintiff concerning this matter, except on cross examination they asked plaintiff's witness if during all the time since 1933 that he had been with the doctor any such practice had prevailed, or any such operation, and he said no. The undisputed testimony therefore is that now to go back to some remote time for them to prove the matter of the experiments with the goat glands would be too remote here. It is a question of his practice now.

THE COURT:—I don't think so. They have charged Dr. Fishbein charged in this article that this man was a quack, engaged in quackery, and I think they are entitled to show anything with regard to his practice that will throw light on the matter. Overrule the objection.

Mr. Morriss Sr.:—If it is not too remote. Note the exception.

THE COURT:—A man that comes into a libel suit practically puts his entire life in issue.

Mr. Morriss Sr.:—We don't think that is correct, your honor.

THE COURT:—It is a question of his reputation. If it is damaged, how much it is damaged. Overrule the objection.

Mr. Morriss Sr.:—If it is a question of reputation, we make the added objection that that is too remote and must be proved also by general reputation and not by specific acts.

THE COURT:—All right.

Mr. Morriss Sr.:—Note the exception.

Q.—When did you first do the operation of taking a testicle out of a young goat and putting in the testicle of a man? *A.*—1917.

Q.—And how long did you continue to do that operation? *A.*—Up until the end of 1932, beginning of 1933.

Q.—When did you first open, when did you move to Milford, Kansas, and start practicing there? *A.*—1917. Yes, 1917.

Q.—1917? *A.*—Yes.

Q.—Did you advertise by pamphlets, newspaper advertisements, or any other way before you started broadcasting? *A.*—Yes.

Mr. Morriss Sr.:—Will your honor note our exception goes to all this?

THE COURT:—If you want to make an objection you had better make it.

Mr. Morriss Sr.:—I hate to interrupt.

THE COURT:—It is your province to interrupt.

Mr. Morriss Sr.:—Anything back of—I mean back of 1930, we would object to as being too remote. That is the objection we make, the objection to this, it is too remote.

THE COURT:—Overrule the objection.

Q.—You say you started broadcasting; first, in answer to my question, when did you start advertising yourself as a doctor? You said you started broadcasting in 1929. Now, I want to ask you if that is when you started broadcasting, have you advertised yourself as a goat gland specialist before 1929? *A.*—Yes, sir, I advertised myself as a specialist on gland transplantation before 1929.

Q.—When did you start advertising? *A.*—I can't be sure. Somewhere around maybe 1921 or 1922, somewhere around there.

Q.—And when did you build a hospital in Milford, Kansas? *A.*—1917.

Q.—1917? *A.*—Yes, sir.

Q.—Now, in those advertisements did you hold out to the world that the goat gland operation would make a man young sexually again?

Mr. Morriss Sr.:—Just a moment. May it please the court, that would be the same objection, it is too remote, and for the further reason that it doesn't call for the best evidence or wouldn't be the best evidence.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—Exception.

Q.—Now, just a minute. In those advertisements that you issued about the goat gland operation from 1917 to 1929 when you started on the radio, did you hold out to the world that that goat gland operation would make a man sexually young again?

Mr. Morriss Sr.:—Just a minute.

Q.—Or words to that effect?

Mr. Morriss Sr.:—Just a moment.

THE COURT:—I have overruled your objection once.

The Witness:—I don't remember.

Mr. Morriss Sr.:—The record wouldn't show any objection to this question. I don't want to interrupt. I wish we might have it understood that the court would allow us to have an objection to all this same line.

THE COURT:—Well, the court has to have an opportunity to rule on whatever is presented. Under the new rules you don't need to take an exception.

Mr. Morriss Sr.:—If it is a different objection we will make it, but otherwise we would not have to.

THE COURT:—It is your duty to except if you think anything is improper. It is no reflection on you to except, I mean to object.

Mr. Morriss Sr.:—I am not speaking about the exception, I am speaking about the objection. I hate to be interrupting.

THE COURT:—I don't know any way you can avoid it. It is the court's idea that these parties are entitled to go into this man's professional record, his reputation, both on the issue of any compensatory damages and on the issue of actual damages; also on the question as to the truth of the allegations contained in the article declared on. I don't think they are limited as to time by the last few years.

Mr. Morriss Sr.:—Well, we think there is a reasonable limit.

THE COURT:—For instance, suppose a man has been practicing twenty-five years without a license at all. You could show that if you charge he is a quack, an unethical practitioner.

Mr. Morriss Sr.:—That is the point we want to be heard on, because we think the court is in error on that and that is the very point I want to be heard on.

THE COURT:—State it now. What is it?

Mr. Morriss Sr.:—I will ask co-counsel—

THE COURT:—No, counsel, we have to proceed here in accordance with the rules of the court. I will hear you if you want to be heard, but don't call your co-counsel.

Mr. Morriss Sr.:—He has the authorities on it, but I may say this just as a matter of principle. For instance, we know that there is always a limit in time upon which you may inquire. It is a matter, as the court said a while ago, which relates to a question of character or reputation. For instance, we know that if you had a witness on the stand in any criminal case you may in criminal cases in Texas inquire about indictments and such as that within a certain period, but that is limited. If it is more than seven years ago it is too remote, it is too much against the presumption of the law that a person has changed in that respect, and here this charge against him is that he is doing, he is now guilty of this, that and the other, and of course to go back to the days of experimentation would not be proper if they are remote. For instance, I might have tried and lost a case or done most any matter in the way of error, or I might have been incompetent, we will say, and then I might have gained competency in the practice of law, and it is the same way with a doctor, the same way with any and every professional man; and so it isn't proper to go back to a remote time to ask about this here, and moreover, here are the specific things that are charged in this libel, certain statements they made in this article are charged upon, many others are not. They have to be confined to a reasonable period of time and to—and of course—I started to say this, that the proof of the truth of any other allegation doesn't help, I mean any other accusation in the article doesn't help upon this. I do not have in mind right now the style of the United States Supreme case where there is a comment on the fact that it is an old custom to try, and certainly a wholly unwarranted one, to try to prove the truth of certain things in the article in justification when the matter to be proved, the truth or falsity is the particular portions of the article that are charged. It doesn't matter that he may have told the truth in other parts of the article if he then slanders or libels him, any other parts of the publication go out, and those that are charged upon and nothing else are the ones; so we say that the lapse of time is such that that is too remote and therefore—and that moreover that isn't a part of the charge in this case, and to prove that doesn't disprove the libel which is sued on here, or to adduce testimony with regard to it, and therefore we say that is calculated to simply prejudice the rights of the plaintiff and all that testimony we will say it is calculated to prejudice the rights of the plaintiff, and it is irrelevant, immaterial and incompetent to establish or disprove the allegations in this case, and entirely too remote.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—Note the exception.

Q.—Doctor, I will ask you that question again, if you please? *A.*—I don't remember that I ever sent out any literature specifying I would make an old man young or anything like that.

Q.—Did you say in any literature that the goat gland operation would be an aid in impotency? *A.*—Very likely I did.

Mr. Morriss Sr.:—Now he is asking again what he said in literature, and the literature is the best evidence.

Mr. Brown:—This is the man that had it put out.

Mr. Morriss Sr.:—That doesn't relieve it.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—Note the exception.

A.—I can't remember the contents of the literature I mailed out years ago, it would be utterly impossible for me to testify to its contents.

Q.—But you do remember, don't you, if you please, in advertising the goat gland operation to the world for ten years, up until 1929, say, when you started on the radio, isn't it a fact you told the people of the United States that if they would let you operate on them with a goat gland it would aid impotency?

Mr. Morriss Sr.:—The same objection, your honor.

THE COURT:—Overrule the objection.

A.—Yes, I think I recommended the implantation of goat, sheep, monkey glands, as an aid to sexual weakness.

Q.—You did that in numerous pieces of literature you put out up to the time you started broadcasting in 1929, did you, Doctor? *A.*—Very likely thousands of them.

Q.—It is the fact you did, didn't you, please? *A.*—I mailed out a lot of literature advertising this gland work I was doing, running into thousands of pieces of literature.

Q.—And you got back thousands of letters through the mail, didn't you, asking about it, before you started on the radio? *A.*—I received a lot of mail, yes, sir.

Q.—Now, you quit the goat gland people, as I understand it—when did you quit fooling with the goat gland proposition?

Mr. Morriss Sr.:—We object to that—when did you quit fooling.

Mr. Brown.—I beg your pardon.

THE COURT:—I think the form of your question has an air of levity.

Mr. Brown:—I beg your pardon.

Q.—When you discontinued the use of the goat gland operation, when was that? *A.*—1933.

Q.—1933. Now, from the time you started in in 1917, up to 1933, how many of those operations, about, did you perform? *A.*—Several thousands of them.

Q.—Did you make people pay up money for it, charge them for it? *A.*—No, the—

Q.—I don't mean— *A.*—The gland tissue was an adjunct to something else.

Q.—When you used the goat gland operation did the patients pay you for that work? *A.*—I didn't sell them an operation for a gland operation alone. The gland was used in conjunction with other work I was doing, as an adjunct to other surgery.

Q.—Did you charge them for that? *A.*—They paid for the glands and other work at the hospital, it was all together paid for.

Q.—Did your literature say that the four phase operation, at the time you had the goat gland operation, that the whole operation, and for the guaranty, you charged them $750? *A.*—That is right.

Mr. Morriss Sr.:—Just a minute. We are going to make an objection and move that that be excluded, for all of the reasons we did before.

THE COURT:—Overrule the objection.

Q.—Well, you say several thousand you operated on. What is your best recollection as to whether that means two or three thousand or five thousand? I don't know what your best recollection as to how many people you operated on when you used the goat gland operation is.

Mr. Morriss Jr.:—Now, of course, we just have to object to questions that pertain to the goat gland operations.

THE COURT.—Well, if you think it improves your situation any do it.

Mr. Morriss Jr.:—Well, we object to it, if the court please.

THE COURT:—It seems to me that you have a good many objections to this kind of evidence. If there is any merit in one, one is just as good as forty, so far as the record is concerned.

Mr. Morriss Jr.:—We don't want to be put in the attitude of waiving by the failure to object.

THE COURT:—I have tried to indicate to you the court's view about the matter. This article, you yourself read all of it, and if you haven't read it all the court would have let the defendant read it. He is charged there with a good many more things than you declare on. They go way back into his history in that article and charge him with matters, and those matters are pertinent and relevant to matters now being inquired about, and when he alleges he is an ethical practicing doctor and his reputation is being damaged and he has been injured, he puts that in issue, and they are entitled to inquire into it. Now, that is my idea, and that is why I rule the way I do, and I tried to make it clear to counsel. Now, if you want to persist in making these objections it is your duty to do so, if you think it will help, but it isn't helping the trial along any.

Mr. Morriss Jr.:—We just want it understood we are not waiving our objection to all of this line of testimony.

THE COURT:—You can state that in the record, but the court isn't making any agreement with you.

Mr. Morriss Jr.:—We want it understood, then—

Mr. Brown.—No, we don't like to appear to be permitted by the word understood.

THE COURT:—I am not making any agreement at all.

Mr. Morriss Jr.:—Then we will have to object, and we object to this as being too remote.

THE COURT:—Overrule the objection.

Q.—You said you had operated on several thousand people using the goat gland operation?

Mr. Morriss Jr.:—The same objection.

Q.—Will you please give us a better idea as to what you mean by several thousand? *A.*—Well, I have in mind five or six thousand people.

Q.—Five or six thousand people? *A.*—Yes, sir.

Q.—And during those years when you were sending out that literature you sent out thousands of pieces of literature, didn't you, telling people that that goat gland operation, in connection with the operation, was a great success?

Mr. Morriss Jr.:—Now, we object to that because any literature would be the best evidence, and that it is immaterial and irrelevant to any issue in the case, and too remote.

THE COURT:—Overrule the objection.

A.—Yes, I told them that the operation was a great success, yes, sir.

Q.—And after using it from 1917, up to 1933, why, you don't use the goat gland technic any more, do you? *A.*—It isn't necessary to transplant glands any more, because we have commercial glandular preparations that we can buy on the market and inject to take the place of the glandular transplantation.

Q.—The question was you don't use the goat gland operation, haven't used it since 1933, have you? *A.*—That is right.

Q.—Now, when you started broadcasting in 1929—by the way, before you started advertising as a doctor were you a member of the American Medical Association? *A.*—Yes, sir.

Q.—And then isn't it a fact that the ethics of that association are opposed to a doctor advertising himself and saying he is the best doctor and that he can cure you? In other words, the ethics of the American Medical Association are opposed to a doctor advertising himself, isn't that correct?

Mr. Morriss Jr.:—Just a moment. We object to that as calling for a conclusion and opinion of the witness, and not being the proper standard or criterion of what would be proper in the case.

THE COURT:—Overrule the objection.

A.—My understanding is that the ethics of the American Medical Association are opposed to any form of advertising by any physician.

Q.—Yes, sir. Now, after you started broadcasting in 1929, and that was when you operated, then, in 1929, 1930, 1931, 1932, and quit in 1933, the goat gland operation, did you tell the people on those broadcasts during those years, did you tell them about the goat gland operation? *A.*—No, sir, I didn't.

Q.—Not over the radio? *A.*—No, sir, I never said anything about the goat gland operation over the radio on none of my broadcasts.

Q.—During the years 1929 to 1933, when you were still using it, did you still put out literature? *A.*—Yes, sir.

Q.—Telling about the goat gland operation? *A.*—Yes, sir.

Mr. Morriss Jr.:—The same objection.

THE COURT:—Overrule the objection.

Q.—In other words, you advertised yourself and the goat gland operation in printed advertisements, is that right, up to 1933?

Mr. Morriss Jr.:—Same objection, if the court please.

A.—Yes and no. There is some period in there, I don't remember just when it was, when the earlier literature, as I remember it, I am just trying to give you an idea—

Q.—That is the literature you put out? *A.*—I know what you are driving at, and I am trying to help you out.

Q.—Well, I thank you very much. *A.*—I am not trying to hedge at all.

Q.—All right. *A.*—The earlier literature was built around a gland operation and the gland operation was, we will say, the star of the literature, and then the gland operation was minimized and the prostate work for enlarged prostates began to become the star in the literature to where the gland operation in the literature began to take a back seat, if you will pardon me for such a homely illustration.

Q.—It had the front seat for ten years or more, didn't it? *A.*—No, not ten years, it didn't have the front seat that long.

Q.—What did you say in the literature from 1929 to 1933, when you stopped using the goat gland operation, what did you say in the literature about the goat gland part of it? *A.*—I don't think I said very much about the gland operation at all in the literature. If I had one of the little blue books I used to mail out we could look at it and see.

Q.—Is this the one about your health—is this the one you mean? *A.*—Let's see if this is the one I am trying to think about. There is something that I have in mind here that I was trying to point out to you. I think this is what I am getting to.

Q.—Is this the one? *A.*—Here is what I am getting to. You see here is the book I am thinking about where all I say about the glands, I discuss it here on page 25 to 26. Gland transplantation, the compound operation is the outgrowth of experiments.

At this point Dr. Brinkley identified the various pamphlets which he has been using from time to time, including those sent from Kansas, pamphlets entitled "Rejuvenation," "The Compound Operation or the Modern Passport to Successful Rejuvenation," the pamphlet entitled "Recharge Your Batteries," the advertisement headed "Every Man Past Forty Should Know the Truth," the pamphlet entitled "The Brinkley Hospitals," the book entitled "The Life of a Man," postal cards used in circularizing patients, also advertisements which had been published in various newspapers.

The testimony then proceeded as follows:

Q.—Doctor, are you the most learned physician in the United States? *A.*—No, sir, I don't think so.

Q.—Haven't you claimed to be the most learned physician in the United States? *A.*—No.

Q.—Haven't you claimed to be the greatest surgeon in the world? *A.*—Maybe some people writing for me have, but I haven't myself.

Q.—Well, this chapter in your book here, isn't it a fact chapter nine is entitled "The most learned doctor in America"? *A.*—It may be. It may be what Mr. Wood put in it, that biography.

Q.—With the copyright held by your wife's sister? *A.*—That is Wood's conception of it.

Q.—Isn't it a fact that on page ten here, or page 110, it says, speaking of you, "Dimly he had begun to realize that he was gifted beyond the run of doctors." Isn't that in your book? *A.*—Maybe, I don't remember.

Q.—And here at page 200, doesn't it say "I'm going to show the American Medical Association, and the doctors and people of this country, that John R. Brinkley has more medical knowledge than any of them have shown"? *A.*—I never said it. Probably Wood did.

Q.—You are going to disclaim everything that appears in your biography? Are you or not? *A.*—I am—

Mr. Morriss Jr.:—We object to that as being argumentative.

Mr. Brown:—I withdraw that question, if the court please.

Mr. Morriss Jr.:—His biography—

THE COURT:—The question is withdrawn, counsel.

Q.—Isn't it a fact on page 203 you said you are going to show "to the jealous organized doctordom in America, that he was more qualified than any of them"? *A.*—No, I never said that. It may be in the book. Wood may have said it, but I never said it.

Q.—Page 214, doesn't the book say here, "in his presentation of facts, no matter on what subject, he is a student of human nature, a psychologist, a master showman, as well as one of the world's most learned doctors and surgeons"? Isn't that in your book?

Mr. Morriss Jr.:—We object to counsel continuously putting into the question "your book."

THE COURT:—Let's find out something more about the book, its connection.

Mr. Morriss Jr.:—It is composed and written by another person.

Mr. Brown:—No, I asked—

THE COURT:—Let's find out whether it was published under his authority or not, whether he knew about it, whether he ever repudiated it or anything. You haven't gone far enough with it.

Q.—Is there a new edition of this book, a later one than this? *A.*—Yes, sir.

Q.—When was the latest one published? *A.*—I declare I don't know. About two years ago, I think something like that.

Q.—Has it got a blue back on it, has it a stiff back? *A.*—It has a cover on it something similar to this.

Q.—Well, hasn't it got a stiff back like an ordinary book? *A.*—No. The one with the blue back is the first edition.

Q.—Oh. *A.*—That is the first one.

Q.—That is the one which is stiff like an ordinary book? *A.*—Yes, bound in cloth.

Q.—Where did the man get the facts from that wrote this book? *A.*—Well, he collected them up from different places. I employed him to write a biography of myself.

Q.—I see. *A.*—And I don't know, he asked me and he asked my wife; he asked people that were acquainted with us. He went back to North Carolina where I was born and you might say made a research of me, and then he wrote this book.

Q.—Have you ever repudiated this book? *A.*—No, I haven't repudiated it.

Q.—Did you pay him for his work? *A.*—Yes, sir, I paid him to write it.

Q.—Did you read any of it? *A.*—Yes, sir.

Q.—Did you read all of it? *A.*—No, I don't know as I have read all of it, but I have read a lot of the book.

Q.—And you hired, you employed him to write it for you? *A.*—That is right.

Q.—And you paid him for writing it? *A.*—I did.

Q.—And you have had how many editions issued, altogether? *A.*—Three or four, I think about four editions.

Q.—And you are selling the latest edition of it right here in Del Rio now, aren't you? *A.*—No, sir.

Q.—Who is selling the latest edition? *A.*—Nobody.

Q.—It is out of print, is it? *A.*—No.

Q.—Where could we get the latest edition? *A.*—You would have to get them from the printer, I guess, in Kansas City, Missouri. We don't have any of them on hand here.

Q.—Sir? *A.*—I say we don't have any of them on hand here in Del Rio, because we don't do any mailing from Del Rio, you see.

Mr. Brown:—Your honor, I think that connects it sufficiently.

THE COURT:—How has the book been distributed?

Q.—Who has sold the book? *A.*—I gave it away, what was distributed. I never sold the book. The first edition—

THE COURT:—You sent it out as an advertisement?

The Witness:—No, I just sent it out to people to let them read it. A lot of people write to me and ask me a lot of questions.

THE COURT:—All right, go ahead and ask him your questions. I believe the last matter before the court was counsel's objection to your asking him about what was in the book. I overrule that objection.

Q.—Doesn't it say in the book on page 310, quoting the "splendid words of Sidney Lanier: 'What possible claim can contemporary criticism set up to respect—that criticism which crucified Jesus Christ, stoned Stephen, hooted Paul for a madman, tried Luther for a criminal, tortured Galileo, bound Columbus in chains, drove Dante into the hell of exile . . . and committed so many other impious follies and stupidities that a thousand letters like this could not suffice even to catalogue them?' Of their company is John R. Brinkley." Isn't that in your book on page 310? *A.*—Probably is, yes, sir.

Q.—Yes, sir. Doctor, I want to ask you now about some things the book says about the goat gland proposition, about what you claim about the goat gland proposition, or rather operation, as to whether it includes sexual powers, that is what I want to ask you about. Now, on page 95, isn't it a fact that the book says, in speaking of the goat gland operation, when you first considered, "it was such an easy field, he knew, for any practitioner to hold out false hopes to a troubled patient, and charge him almost anything. Other doctors had told him how the suckers bite, at any bait, when they are troubled in this vital aspect of their own manhood." Is that in your book at page 95? *A.*—It very likely is, yes, sir.

Q.—Were you the first man that ever did this operation of taking the goat testicle and putting it in the man's testicle? Were you the first man that ever did that? *A.*—So far as I know I was, yes, sir.

Q.—Does your book state here at page 101—you remember you operated on two old farmers first, didn't you, two old farmers?

Mr. Morriss Jr.:—We make the same objection.

Q.—At Milford, Kansas—

THE COURT:—Overrule the objection.

Mr. Morriss Jr.:—It is too remote, irrelevant, immaterial and a specific instance rather than a general matter.

A.—The first man I operated on was 45 years of age, and I think the second man I operated on was about 38 years of age.

Q.—Were they troubled with loss of sexual power. *A.*—Yes, sir.

Q.—What effect did the goat gland operation have on them? *A.*—They claimed complete restoration of their sexual powers.

Q.—Then did you operate on two people kind of having trouble with their minds? *Y.*—Yes, sir.

Q.—And the fifth operation, was that on a boy when you passed a barber shop and heard them making fun of the operation? Did you take a young man out and operate on him, or rather did you operate on one of the boys that was there in the restaurant or barber shop? *A.*—I think the third operation was a cashier in a bank that went insane.

Q.—I say you had two farmers and then two that were troubled in the mind, and was the fifth one a man around Milford, when they were kind of laughing about the operation? *A.*—Oh, I know who you mean, old Charley Tasine. The boys in the barber shop were kidding Charley, whether he would have an operation or not, and he said he would if he had the money, he would have an operation, and I was coming along, and I said, "You don't have to have any money, come up to the hospital and I will give it to you for nothing."

Q.—And you gave it to him? *A.*—Yes, sir.

Q.—You took him up and the operation was entirely successful, wasn't it? *A.*—Yes, sir. He was a bachelor and he got married right afterwards, anyway.

Q.—Didn't the book say on page 101 that "the man to whom Doc had done this had become a regular billy goat, twice as good as any other man around Milford"? *A.*—That is what he claimed. He was one of these boasting fellows, liked to blow off as to his ability.

Q.—About that time you owed some money on your hospital, didn't you? *A.*—Yes, sir.

Q.—And you hired a publicity agent to come down there and help you advertise, did you? *A.*—I hired a man that had had experience in advertising to come down there and advise me if there was anything I could do to increase my business.

Q.—Yes, sir, and didn't he ask you, "Well, you are just a country doctor here away out in the country. What has happened here unusual, what has been done here that we can advertise?" Didn't he ask you about that? *A.*—Yes, he did.

Q.—And finally after your studying around about it didn't you tell him about this seven, I think it was, operations you had performed with the goat gland operation, or whatever number it was? *A.*—I don't remember whether I had seven at that time—

Q.—I don't mean the number, if you will excuse me, but didn't you tell this advertising man finally, "As far as doing anything different, I will tell you, I have done this goat gland operation"? *A.*—That is right.

Q.—And didn't he reach over and grab you by the hand and say "Well, we have them, it is worth a million dollars"? *A.*—He said, "Dr. Brinkley, you have something that is worth a million dollars to you."

Q.—Well, it has been worth a whole lot more than that, hasn't it, Doctor?

Mr. Morriss Sr.:—That is irrelevant and immaterial.

Mr. Brown:—We say he has been getting the shekels out of the people.

THE COURT:—I think it is a matter of argument. You were asking about the goat gland operation. Sustain the objection.

Mr. Brown:—You sustain the objection?

THE COURT:—To that question, yes.

Q.—The book here on page 155 quotes from you; it says "I began removing several inches of the vas deferens, lymph chain and vas deferential artery, together with a crown suture through the pampiniform plexus located in the epididymis; and in addition to this surgical technique the transplantation of the testicles from the three-weeks-old Toggenberg goat. Needless to say, the results were astonishing, miraculous." That is in your own words, isn't it, Doctor? *A.*—Yes, sir, I have seen astonishing and miraculous results, yes, sir.

Q.—Now, I don't mean to go into technical details of it. Let me ask you this. I may have it wrong in my mind. *A.*—All right.

Q.—Is it a fact that in operating on the goat gland operation, as I understand it, and please correct me if I haven't got it right, you didn't remove any of the testicles out of the man, did you? *A.*—The ordinary goat gland operation is what you are thinking about, isn't it?

Q.—Yes. *A.*—Certain ones I took and cut a hole out of the man's testicle and took a chunk out and filled the hole up in the testicle with goat gland.

Q.—Ordinarily you would cut a little flap or slip? *A.*—Just a pocket to put the gland in.

Q.—Would you put a piece of the goat gland in that little hole? *A.*—No. A 3 week old goat, I would use a 3 weeks old gland, just take the capsule off of the gland and transplant the whole gland.

Q.—You would put a goat testicle in one man's testicle and another in the other side? *A.*—Yes, and sometimes put them in the abdominal muscles. There were different locations for them.

Q.—Now, Doctor, we say in this piece that you are suing us about here, one of the things that you are suing us for, it says that the effect of it is that since you started advertising the goat gland operation that you have made lots of money. That is a fact, isn't it? *A.*—I think that is something we all have to argue about, whether a fellow makes lots of money or not.

Q.—I don't mean whether you have spent it or spent it on expenses or used bad judgment as to what you did with your money, but this says this, it says "he continues to demonstrate his astuteness in shaking shekels from the pockets of the credulous Americans." In other words, that is one of the things you are suing us about, is whether you have gotten lots of money out of the American people, isn't that a fact? *A.*—Well, it is the way you state it; shaking shekels is entirely different, to my way of thinking, a different expression than "you have made a lot of money by operating on people."

Q.—All right, over here there is another place that they allege on where it says it has been reported that at some times you have made as much as $55,000 a week. "The evidence assembled indicates that at various times Brinkley has made as much as $55,000 a week as a result of his various quackeries." *A.*—I never made anything like $55,000 a week. I wish I had.

Q.—You stated that you operated on five or six thousand for the goat gland operation, and the main operation was $750. *A.*—Yes, but only a small per cent pay $750.

Mr. Morriss Jr.:—We object to going into that. It is irrelevant and immaterial to any issue in the case, as to what was made out of it or what profit was made out of that, and we make that additional objection.

THE COURT:—I think the authorities all hold that the entire article is admissible. If you can cull certain parts out and say they are false and libelous, they have the right to put in the whole article for the purpose of seeing the drift and purpose of the entire writing.

Mr. Morriss Jr.:—Yes.

THE COURT:—And while the fact part may be true wouldn't relieve them from responsibility for that part which is false, still in an effort to show the article is substantially or wholly true, I think they are entitled to take it up entirely. Overrule the objection.

Mr. Morriss Jr.:—If the court please, we think these matters, other matters, unless they reflect light upon the particular part charged on, are improper to be gone into.

THE COURT:—I know, but the question is the damage this man suffered, that is in the case, and you can't compress it into any narrow confines and get a just trial, and I think that they have put him on the stand and that allows them to do it. They are entitled to go into the matter very fully and ventilate it if they want to, so I am going to overrule the objection.

Q.—Doctor, I am not asking you now about your net income report or your report to the government or anything of that kind, but I want to ask you what was the gross income, the total income of yourself and your wife for the year 1938?

Mr. Morriss Jr.:—We object to that as being immaterial and irrelevant and improper and incompetent.

THE COURT:—Counsel, I ruled on your matter. You and I have an entirely different conception of the ruling with regard to preserving your record. I think if you make it clearly known to the court that you object to a certain line of procedure and the court overrules you and announces that that line of procedure is to go on, you have done enough. You seem to think you have to get up and make an objection every time a question is asked. I think you had better look at that new rule.

Mr. Morriss Jr.:—I think this was a different question.

THE COURT:—The point about it is, it is militating against the trial in two ways. In the first place, it is taking a lot of time and, in the second place, it is so breaking up the thread of what is sought to be brought out that the jury and court and everybody else loses the natural continuity of the story, and I am going to ask you to consider that rule and see if you think it is necessary for you constantly to do this. I know the nature of your objection. I told you what the court's ruling is. I have gone to the trouble several times to outline to you my idea as to what I think the course of the trial ought to be. If it is contrary to your idea, why, after having stated your reasons, don't you let it go at that? The record is going to show what has transpired. I don't think it is necessary for you to interrupt every time anybody does anything.

Mr. Morriss Jr.:—If the court please, we thought this was a different line of questioning, and we think it is because, in the first place, as I understood counsel, it related to 1938. Is that correct?

Mr. Brown:—Yes, sir.

Mr. Morriss Jr.:—Which was subsequent to the time of the article.

THE COURT:—I don't see the difference. I ruled they could inquire into the money he made out of these operations that were a part of his advertising campaign. The mere fact they confine it to one year doesn't change the ruling. I am going to have to ask you to help the court a little bit. I don't want to appear to be querulous or arguing with you, still you will have to assist in the trial of this case. Otherwise we will be here too long.

Mr. Morriss Jr.:—We were under the wrong impression a while ago.

THE COURT:—I know, but there doesn't seem to be any way to get rid of you. Every time I say something it provokes a new argument out of you.

Mr. Morriss Jr.:—Well, I didn't want to be put in the attitude of waiving any matters, and secondly, with reference to this, we thuoght it was an entirely different matter and inquiries as to his general income.

THE COURT:—That is the fifth time you told me it was a new matter. If you think that is helping us to go forward, I don't see it.

Mr. Morriss Jr.:—We except to the court's remark.

THE COURT:—All right.

Q.—What was the gross income of yourself and your wife for the year 1938? *A.*—Our gross income for the year 1938, from all sources—I can't give you the exact figures because my auditors make this out—is around $810,000 gross.

Q.—Yes, sir. Now, what was your gross income for the year 1937? *A.*—If I remember correctly, the gross income for 1937 was pretty close to eleven hundred thousand, right at $1,000,000 gross. Either it was a little over ten hundred thousand or a little under, I just can't remember those exactly, but I have it, of course, at my office in Little Rock. I have copies of my income tax return. That would give the dollars and cents.

Q.—You took a trip one time on a boat to China, didn't you, you and your wife? *A.*—That is right, yes, sir, in 1923.

Q.—And you had had a great deal of unfavorable publicity in the papers before you took that trip, hadn't you?

Mr. Morriss Sr.:—Just a moment. We think—

THE COURT:—I think the question is argumentative.

Mr. Morriss Sr.:—Besides, it refers back to a time in 1920 or '21. I believe he said. It is very, very remote.

Q.—It is a fact that before, just before you took that trip and while on that trip you had a great deal of very unfavorable comments in the newspapers, but—

Mr. Morriss Sr.:—Just a moment. If so, that would be hearsay and not binding on this plaintiff.

THE COURT:—You can ask him if he had been commented on in the newspapers just before he took the trip. I don't know what you are leading up to.

Mr. Morriss Sr.:—Let it be understood that we object to it primarily because it is too remote and not an issue in this case and collateral, immaterial and irrelevant.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—And hearsay, of course.

THE COURT:—Overrule the objection.

Q.—It is a fact, is it not, that before you took that trip and when you were on that trip the newspapers commented very widely on you in an unfavorable way? *A.*—No, sir, I don't think that happened until after I was in China. I think it broke loose after I left.

Q.—Doesn't the book say it was before and the passengers even shunned you on the boat? *A.*—Oh, Lord, no. Oh, no, the comment about the passengers on the boat was we were guests of the captain of the steamer and there was a certain amount of jealousy due to that.

Q.—You tell about full dress dining with the captain, don't you? *A.*—Yes, sir.

Q.—Doesn't it say on page 166, "All aboard!" And then it says that "at the very time when he was completing his plans to go to Shanghai, at Dr. Thompson's invitation, to operate on wealthy Chinese sufferers"? And then it says "These flaming headlines in every paper against him made it practically impossible for him to secure patients in America." Don't you remember that those newspaper attacks were on you before you left for China? *A.*—No, I don't think they really got started until after I got over there. I don't think they had.

Q.—All right. Excuse me. *A.*—I ought to have—I am not trying to dodge the issue, but that is my impression. You will probably have copies of those newspapers, and it will give the dates.

Q.—I just read this part out of the book. Doesn't it say on page 157 "Unfortunately, the provincial-minded Americans on board were jealous at these attentions, and left the Brinkley party pretty much to itself"? *A.*—That was on account of the captain being so solicitous to Mrs. Brinkley and setting aside reservations in the picture show, and he ignored the rest of the passengers, and I think they resented so much attention being paid to us by the captain of the ship.

Q.—Now, as to this article in the *Hygeia*, published by the American Medical Association, it is a fact, is it not, Doctor, that the newspapers and magazines of this country on several occasions have commented most unfavorably about you and your work?

Mr. Morriss Jr.:—We object to that as a conclusion, calling for a conclusion and opinion of the witness, and would not be the best evidence and is irrelevant and immaterial and incompetent to establish any issue in the case, and hearsay.

THE COURT:—This is cross examination here, in effect, under the rule. Overrule the objection.

Mr. Morriss Jr.:—Note the exception.

A.—I think that what adverse criticism that I have read in the magazines and newspapers concerning me has probably had its basis in statements from the American Medical Association, which they used as an authority in denouncing me.

THE COURT:—Doctor, he didn't ask you that question.

Q.—I asked you isn't it a fact that at different times up to the time that this article was published the newspapers and press of the country have attacked you very severely, you and your work? *A.*—I don't think the press of the country knows it. The Kansas City *Star* put on—

Q.—I am just asking you, if you please, about the press of the country. You can say no if you like. *A.*—I would say no to that, that is the way I would answer that.

Q.—Do you remember what they call the diploma mill scandal? *A.*—Yes, sir, I do.

Q.—Isn't it a fact that the newspapers of the country in this diploma —what do you mean by diploma mill scandal? What is that, please? *A.*—It refers to a situation that was existing pretty much all over the United States, and I believe its headquarters were in the state of Missouri. There was some understanding between certain people operating medical schools in the state of Missouri and secretaries of state boards of the various states where that somebody that didn't have the proper qualifications or something like that would buy or get a degree from one of these schools and then by some aid from somebody get by the board.

Q.—In other words, they would get their diploma without studying like they are supposed to do, but by paying in some way, they would get a diploma when they were not entitled to it? *A.*—Some hocus-pocus, yes, sir.

Q.—That is what you called the diploma mill scandal, wasn't it? *A.*—Yes, sir.

Q.—And that was pretty widely discussed throughout the country in the newspapers, wasn't it? *A.*—Yes, sir.

Q.—And in connection with that didn't the newspapers charge you, that you had gotten your license and your diploma in this diploma mill route, that they turned out the diplomas like a mill ground them out?

Mr. Morriss Jr.:—We object to that. It is not the best evidence and is hearsay, calls for a conclusion and opinion and not competent to establish any issue in the case and as being too remote and is an improper attempt to show reputation by specific, any specific instance, and show truth by hearsay.

THE COURT:—What do you say about that, counsel?

Mr. Brown:—Well, your honor, just in mitigation I want to show that at former times during his professional career—he is complaining of damages by reason of this article, and certainly we have a right to go into whether the other papers have commented on his standing, in mitigation of the damages.

THE COURT:—All right, overrule the objection.

Mr. Morriss Jr.:—Note the exception.

Q.—Isn't it a fact that you became branded from end to end of the land as a diploma mill graduate?

Mr. Morriss Sr.:—Just a moment. If the court please, we make the same objection for each and all of the reasons just now given, and the inquiry is not directed as to any particular time and it is in no way shown that it is or could in any way be proved in mitigation, as counsel said was the purpose, of damages in this case because of the remote time and that some other paper may have done, and of course that further calls for a conclusion and is argumentative.

THE COURT:—If these facts be true wouldn't they be entitled to be considered with regard to the motive and intent of the defendant Fishbein in making these charges, as to whether or not he had grounds upon which he honestly believed them?

Mr. Morriss Sr.:—I shouldn't think for a moment—

THE COURT:—The authorities have held that.

Mr. Morriss Sr.:—I shouldn't think for a moment a publication back in 1920, we will say, whether it was right or wrong, whether slanderous or not, by somebody else could be put in evidence to mitigate damages for a libel published in 1938. I wouldn't think so, your honor.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—We accept.

At this time a short recess was had, at the conclusion of which the following proceedings were had, in the presence of the jury:

THE COURT:—All right, read the last question.

The reporter thereupon read the last question, as follows:

"*Q.*—Isn't it a fact that you became branded from end to end of the land as a diploma mill graduate?" *A.*—I couldn't say that I was branded from one end of the land to the other as a diploma mill graduate. I know the newspapers carried a lot of articles about me being connected with a graduate of that school.

Q.—That they called a diploma mill? *A.*—Yes, sir.

Q.—And those pieces appeared all over the country in papers, didn't they, Doctor? *A.*—I am quite sure many, many papers carried it, yes, sir.

Q.—Isn't it a fact that Henry Ford's Dearborn *Independent* carried a lengthy attack on you and he called you the dean of quacks?

Mr. Morriss Sr.:—The same objection, may it please the court, the same objection.

THE COURT:—Overrule the objection.

Mr. Morriss Jr.:—At the same time we make—

THE COURT:—Please observe the rule as to only one counsel.

A.—In 1923—

Mr. Morriss Sr.:—Of course, we make the motion as to this and all matters that have preceded it on this question wherein the attempt to get that in under the guise of mitigation, we make the motion first that it be excluded and the jury instructed not to regard it for any purpose, for all of the reasons that we have stated an objection to it, and secondly, that as to each and all of it that it be limited and the jury be instructed so that they may understand that it is limited to the purpose of mere consideration, and cannot be had for any other purpose on this than the subject of mitigation.

THE COURT:—Well, I think it is admissible not only from the standpoint of mitigation but it is admissible as going to the good faith of the defendant in the matter, as to whether he actually believed what he was writing.

Mr. Morriss Sr.:—Then limited to those purposes—

THE COURT:—If you want it limited that way the court will limit it that way.

Mr. Morriss Sr.:—We want it limited only in the event the court overrules our objection, which we consider good, and admits it.

THE COURT:—I will overrule the objection and limit it to those purposes.

Q.—Dr. Brinkley, the Kansas City *Star*, what town is that published in, Kansas City, Kansas or Missouri? *A.*—Kansas City, Missouri.

Q.—And what has the daily circulation of the Kansas City *Star*—what is that circulation, do you know? *A.*—I think it was 245,000 at that time.

Q.—When you were practicing there in Kansas the Kansas City *Star* was attacking you in various ways, was it not? *A.*—Daily, yes, sir.

Mr. Morriss Sr.:—Just a moment. The same objection, it is too remote for any purpose here.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—That is among all of the others.

Q.—Do you know whether the Kansas City *Star* was read in every state in the union? *A.*—I don't know what their circulation—I don't know where they went to.

Q.—Yes, sir. They would write up these things, put them on the front page, wouldn't they, the Kansas City *Star*? *A.*—Many of their articles were on the front page.

Q.—Now, were the papers at the time of those attacks by the Kansas City *Star* on you, did the papers of the country have headlines about your alleged fakery, quackery and fraudulent activities?

Mr. Morriss Sr.:—Same objection, your honor.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—Also it is a conclusion and hearsay.

A.—For some strange reason the papers over the country didn't seem to copy these articles out of the Kansas City *Star* and reprint them. Very few were printed by other papers. I had a clipping bureau to see what distribution there was over the country of those articles at that time, and it seemed it was confined mostly to the Kansas City *Star* itself.

Q.—Isn't it a fact that the papers all over the country carried pieces about your alleged fakery, quackery and fraudulent activities? *A.*—I suppose many papers did.

Q.—Isn't it a fact, Doctor? *A.*—I wouldn't say a fact.

Q.—I don't mean every paper. *A.*—I don't know the extent. When I say papers all over the country carried it I would be saying every paper over the United States.

Q.—No, I don't mean that, but papers generally? *A.*—No doubt many papers.

Q.—Carried articles accusing you of being a fake and quack and things of that kind? *A.*—I think many papers did.

Q.—Yes, sir. In the American Medical Association—what is that, please, what is the American Medical Association? *A.*—Well, the American Medical Association, I believe, is a corporation incorporated under the laws of the state of Illinois with principal offices at 535 North Dearborn Street, Chicago, and it publishes a weekly medical journal and other publications, including *Hygeia* Magazine.

Q.—How long have they been— *A.*—And they publish a medical directory.

Q.—How long has the American Medical Association been in existence, do you know about how long? *A.*—I don't know whether they were organized in 1856 or 1876.

Q.—Isn't it a fact, Doctor, that most of the regular physicians throughout the United States are members of that association?

Mr. Morriss Sr.:—We object to that as being immaterial and irrelevant to any issue here.

THE COURT:—Overrule the objection.

Mr. Morris Sr.:—Note the exception.

A.—My impression is about 80,000 physicians belong to the American Medical Association, and I think the total number of physicians in the United States is somewhere around 140,000 to 150,000. That is what is in my mind. It may be more than that, because I am not in a position to be an expert on how many doctors there would be. That would be for Dr. Fishbein to say.

Q.—You haven't much opinion for the activities of the American Medical Association, have you? *A.*—In some respects I don't like their activities.

Q.—Do you think most of the men in the association instead of being Christian gentlemen as of old, they are politicians and abortionists, do you think that is what the members of the American Medical Association do?

Mr. Morriss Sr.:—We object to that as being immaterial and irrelevant to any issue in the case, as to what he would think.

THE COURT:—Sustain the objection.

Mr. Brown:—We except to the ruling of the court.

Q.—What was your income in the fiscal year 1936? *A.*—I couldn't tell you that.

Q.—You don't have an approximate idea about it? *A.*—No, I don't remember what it was.

Q.—Whether it was a half million or a million? *A.*—I would have to get the information from my bookkeeper.

Q.—You can't remember whether it was a half a million or a million? *A.*—No, I can't.

Q.—All right. Dr. Brinkley, so that you will understand it, if you please, the first paragraph of the article says this, "He continues—" that you are suing on the part of the article—I will say this so you will understand what I am asking you about in these questions. It says, "He continues to demonstrate his astuteness in shaking shekels from the pockets of the credulous Americans, notwithstanding the efforts of the various governmental departments and agencies." Now, throughout your practice the governmental departments and agencies have tried to stop you from these advertisements, and as far as your license and broadcast is concerned they have been opposed to you, haven't they, the governmental agencies? *A.*—No, sir, not that I can think of.

Q.—That is the way you want to answer it? *A.*—Yes sir.

Q.—All right, sir. Well, you had, at one time you had a license to practice medicine in the state of Connecticut, didn't you? *A.*—Yes, sir.

Q.—The state examining board of Eclectic, the Eclectic board after an investigation and hearing, they revoked your license to practice in Connecticut, did they?

Mr. Morriss Jr.:—Just a moment. We object to that as being immaterial and irrelevant and incompetent to establish any issue in the case, and that any order which was made by the board would be the best evidence and that it is incompetent to establish any issue in the case and would be hearsay and incompetent and prejudicial to the rights of the plaintiff.

THE COURT:—Overrule the objection.

Mr. Morriss Jr.:—Except.

Q.—It is a fact that your license to practice medicine in the state of Connecticut, after investigation that your license was revoked, was it not? *A.*—No investigation was made.

Q.—Well, it was revoked? *A.*—They were revoked, yes, sir, when I was in China. I never was even called before the board or an investigation made. They put out a blanket order and revoked the licenses of every Eclectic doctor in the state of Connecticut, some having been licensed there for fifty years.

Q.—Your license in Connecticut was revoked? *A.*—Yes, sir.

Q.—You had this radio station in the state of Kansas? *A.*—Yes, sir.

Q.—Were you operating that station under permit of the United States government? *A.*—Yes, sir.

Q.—That permit was issued under what they call the Federal Radio Commission? *A.*—At that time, yes, sir; Federal Communications Commission now.

Q.—Now, how long, altogether, did you operate your Kansas station? *A.*—It was built in 1923 and I sold it in 1931.

Q.—When did the federal government revoke your radio license or refuse to renew the license of your radio station in Kansas? *A.*—They refused to renew the license in June 1930.

Q.—June 1930? *A.*—Yes, sir.

Q.—Now, is it a fact that a reporter for the *Star*, the Kansas City *Star*, that this reporter was a great booster of President Hoover in the Republican convention when Hoover was first elected President?

Mr. Morriss Jr.:—I don't see any pertinency, your honor.

Mr. Brown:—I will show it, your honor, in just a moment.

THE COURT:—Unless you can indicate some pertinency to it it seems to be going too far afield.

Mr. Brown:—I want to ask him if he didn't accuse President Hoover himself, after he was President of the United States, of influencing the Radio Commission to revoke his license, showing under our allegation here you have done these things even though the governmental agencies and governmental departments have tried to stop you.

THE COURT:—All right, overrule the objection.

Mr. Morriss Jr.:—Note the exception. We move the court now to instruct the jury not to consider these remarks.

THE COURT:—Yes, the jury will, of course, realize that the remarks of counsel are not evidence. You will not consider his remarks as evidence, it is simply made to assist the court in ruling on the objection.

Mr. Brown:—Yes, sir.

THE COURT:—You will be governed by what you hear from the witness stand. Go ahead.

Q.—What was the man's name, do you remember it? He was a prominent reporter on the *Star* that was one of the first promoters of the—of President Hoover's campaign for the presidency. Do you remember the name of that reporter? *A.*—No, sir, I do not. May I help you out a little bit? I think you are thinking of the managing editor of the *Star*, Mr. Roberts, that was the promoter of Mr. Hoover's campaign. It never occurred to me it was a reporter. I think it was Mr. Roberts, the managing editor of the *Star*.

Q.—He was a great supporter of Mr. Hoover's? *A.*—Yes, sir.

Q.—Then Mr. Hoover appointed some members of the Federal Radio Commission, did he not? *A.*—I guess he appointed them all.

Q.—Yes, sir. *A.*—I think he did.

Q.—Is it a fact that even President Hoover himself, when this controversy came up about refusing your license in Kansas, that he advised the Radio Commission that they ought to revoke your license or influenced the Radio Commission?

Mr. Morriss Sr.:—Now, may it please the court, that would be the rankest hearsay.

THE COURT:—I think you would have to show this witness knew it of his own personal knowledge.

Mr. Brown:—I will show that he has charged that in a document which he has ——

THE COURT:—Then ask him about that, don't ask him this as a matter of original evidence.

Mr. Brown:—Yes, sir.

THE COURT:—Sustain the objection.

Mr. Brown:—Note the exception.

Q.—Isn't it a fact that on page 229 of your book in reference to when they were having a hearing about renewing your license it was stated, "and the *Star*, as the A. M A. and everybody knew, was very close to the new president; and, in turn, he held a position of advantage over the Federal Radio Commission." Didn't you state that in your book? *A.*—Yes, sir.

Q.—And didn't you say "That was the set-up. It was inevitable that the A. M. A. would seek the help of the *Star*. It was inevitable that the *Star* would throw itself enthusiastically into the fight. There are things in history that never see the light of day." McDonald is the name of that reporter I was thinking about. Was there a reporter named McDonald? *A.*—Yes, sir, that was one of their reporters.

Q.—It is a fact, then, that you claim in the book here that the President himself interested himself with the Radio Commission to get your license revoked, isn't that a fact? *A.*—Yes, sir, that was told to me by Vice President Curtis, is where I got my information.

Q.—Vice President Curtis told you the President himself had interested himself? *A.*—Yes, sir.

Q.—And the Radio Commission had a hearing with respect to renewing your license, did they not? *A.*—Yes, sir.

Q.—And the Radio Commission of the United States government refused to renew your license in Kansas, did they, Doctor? *A.*—Three did and two didn't.

Q.—But the majority refused to renew your license? *A.*—Yes, sir.

Q.—Now, you have had a pretty severe contest in reference to the state board or authorities out there in California, didn't you? Didn't you apply for a permanent license to practice in California? *A.*—Yes, sir.

Q.—You practiced there a few weeks under a temporary license, didn't you? *A.*—Yes, sir.

Q.—Made $40,000 there, didn't you? *A.*—I don't recall what the gross returns were.

Q.—Something like that, wasn't it? *A.*—I consider it was something like that, yes.

Q.—Then after you came back to Kansas isn't it a fact that you were indicted in California and that they sent the officers out to Kansas to bring you back to California?

Mr. Morriss Sr.:—Just a moment. The same objection, that is, the objection that it is immaterial and irrelevant and incompetent to establish any issue in this case, doesn't show any character of wrong upon his part or any character of impropriety or unethical conduct, and it is hearsay.

THE COURT:—What character of indictment was that?

Mr. Brown:—Practicing medicine in conspiracy with another fellow, another doctor, contrary to the laws of California.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—And the fact that that happened—in other words, an indictment is no presumption of any man's guilt of anything.

THE COURT:—I think that is perfectly true, counsel, but when you are inquiring into the question as to the intent and motives of a party in writing an alleged libelous article, and when you are inquiring into it not only from the standpoint of a basis of compensatory damages, but you accuse him of malice and ask for exemplary damages, you are entitled to put yourself in his place and see what light and information he had that he thought was true that would warrant him in writing this article, and accordingly you are on a different footing. Overrule the objection.

Mr. Morriss Jr.:—But we do move that it be definitely understood as not being admissible except—

THE COURT:—As not being admissible for the purpose of proving the alleged indictment, but simply on the question of mitigation and the question of the good faith of this party in writing this article. All right.

Q.—Well, that is a fact, isn't it, Doctor, you were indicted in California and they sent officers back to Kansas to try to take you to California *A.*—Yes, sir.

Q.—And you were very much displeased with the activities of the United States government in reference to refusing to renew your radio permit, were you not? *A.*—Displeased?

Q.—Yes.

Mr. Morriss Jr.:—The fact that he was displeased is irrelevant and immaterial.

Mr. Brown:—Your honor, it is under the question of the activities of the governmental agency.

THE COURT:—Whether he is displeased or not, what would that tend to prove? It naturally follows that if he wanted his license renewed and they wouldn't renew it he wouldn't like it, but I don't like the form of your question.

Mr. Brown:—All right the question is withdrawn.

Q.—Isn't it a fact you stated and circulated matter in reference to your contest with the governmental departments—that you state in this book and circulated that California has a legal record for persecution that stinks to Heaven? A.—It probably is in the book, yes, sir.

Q.—"Tom Mooney is still in jail there. The United States has a legal record for persecution that stinks to Heaven." Didn't you put that in about your government with reference to this, and didn't you put that in the book and circulate the book? *A.*—I think it is in there, and the book was circulated.

Q.—There it is—"the United States has a legal record for persecution that stinks to high Heaven." That is in there, isn't it, Doctor? *A.*—Yes, sir.

Mr. Morriss Sr.:—We move the exclusion of that for all of the same reasons and for the further reason it is shown that the article or book is not written by Dr. Brinkley but written by a wholly different man.

THE COURT:—I know, but the doctor said he paid the man to write it. He read it and has been giving it away.

Mr. Morriss Sr.:—He said he read parts of it. He made the statement he never had read it.

THE COURT:—That goes to the weight of the evidence rather than to the admissibility. He is circulating the book, he says he is giving it away. I think that is legitimate examination. Overrule the objection.

Mr. Morriss Sr.:—Note the exception.

Q.—Now, after the Federal Radio Commission refused to renew your permit in Kansas you appealed that case to the Court of Appeals of the District of Columbia, didn't you? *A.*—That is right, yes, sir.

Q.—And that court agreed with the government? In other words, they sustained the action of the board in refusing to renew your permit? *A.*—Yes.

Mr. Morriss Sr.:—Same objection, may it please the court, and for the further reason that nothing of that matter is in the publication which is complained of, nor would it be the best evidence.

THE COURT:—Overrule the objection.

Mr. Morriss Sr.:—Note the exception.

A.—The court held with the commission, yes, sir.

Q.—And didn't you print and circulate in this book here throughout the country here that as far as your contest with the government departments was concerned, that the commission and the court ignored all the weight of the evidence, for they were out to get Brinkley? *A.*—Very likely I said that, yes, sir.

Q.—Now, then, you had a license, of course, to practice medicine in the state of Kansas, didn't you? *A.*—Yes, sir.

Q.—And was it the board of health of the state of Kansas had a hearing— *A.*—No, the board of medical examiners is the licensing body.

Q.—Of the state of Kansas, they had a hearing on whether they would revoke your license to practice medicine in Kansas, didn't they? *A.*—Yes. sir.

Q.—And you had been practicing medicine in Kansas since 1917, up to— *A.*—1930.

Q.—Was it in 1930 that they revoked your license to practice? *A.*—Yes, sir.

Mr. Morriss Sr.:—We make the same objection to all that, your honor.

THE COURT:—All right.

Q.—So this board who licenses people to practice medicine in Kansas, they had a hearing in respect to your case, didn't they, Doctor? *A.*—Yes, sir.

Q.—And after that hearing they revoked your license to practice medicine in Kansas, didn't they? *A.*—That is right, yes, sir.

Q.—Yes, sir, and after they revoked your license you appealed that case to the Supreme Court of the state of Kansas, didn't you? *A.*—No, I don't think it went to the Supreme Court of the state of Kansas. I think it went to some federal court.

Q.—Let me see. *A.*—I don't think it went to the Supreme Court of Kansas, I think it went to the federal court.

Q.—Isn't it a fact that you appealed to the Supreme Court of Kansas after they had revoked your license to practice medicine? *A.*—I don't think we ever went to the Supreme Court of Kansas after the license was revoked.

Q.—Well, isn't it a fact— *A.*—Maybe we did, but it runs into my mind that we went into a federal court.

Q.—I will ask you that in just a minute. Isn't it a fact that on page 244 you say "Meanwhile Brinkley's attorney went before the Supreme Court of Kansas and sought to prevent the hearing"? *A.*—That was before the hearing.

Q.—Oh, excuse me. You claimed that the members of the medical board of Kansas were so prejudiced against you they wouldn't give you a fair trial, isn't that right? *A.*—That is right.

Q.—So you went to the Supreme Court of Kansas trying to have your case heard before somebody else or another board or something of that kind? *A.*—I declare I don't know what went before the Supreme Court, I don't know what we went before the Supreme Court for.

Q.—Anyway, you went before the Supreme Court of Kansas before the hearing? *A.*—Yes, sir.

Q.—And isn't it a fact that in your book here at 244 you copy what the Supreme Court of Kansas said about your case, and didn't the Supreme Court of Kansas say this, or you said this, "The Supreme Court was hardly impartial in its denial of Brinkley's claim, since it went beyond the charges in purporting to express them; holding that they held that Brinkley was 'an empiric without moral sense, and having acted according to the ethical standards of an imposter, the licensee has performed an organized charlatanism until he is capable of preying on human weakness, ignorance and credulity to an extent quite beyond the invention of the humble mountebank' . . . who was 'fleecing the defective, the ailing, the gullible and chronic medicine takers who are moved by suggestion, and is scandalizing the medical profession and exposing it to contempt and ridicule' "? A.—Something like that.

Mr. Morriss Jr.:—We object to that, may it please the court, for the dual reason, first, that it was written in a book which is shown not to have been—he may have authorized it or assented to this man writing his biography and agreed to pay for the publication. That doesn't mean he is the writer or author of it—

THE COURT:—I have already overruled you on that question, counsel.

Mr. Morriss Jr.:—All right. Secondly, the other matter is hearsay, and for the further reason it is simply quoting what some other concern said about him.

THE COURT:—As I understand it, the comment that counsel was just reading is something that the Supreme Court said, is that correct, counsel?

Mr. Brown:—Yes, sir, copied by this man.

THE COURT:—And put in this book and circulated by Dr. Brinkley.

Mr. Morriss Sr.:—Yes, sir.

THE COURT:—Overrule the objection. I can't see why if the doctor put it in the book and circulated it himself he can take exception.

Q.—Now, since you have been here in Texas, isn't it a fact that the state medical board of Texas filed suit to revoke your license to practice in Texas? *A.*—Yes, sir.

Q.—And they filed that suit up in Dallas, didn't they? *A.*—No, here in—yes, I guess it was Dallas. Dallas, yes.

Q.—Now, in respect to this allegation here about the government agencies being against you, has the American government itself, Dr. Brinkley, been against you in your desire to practice medicine and your advertisements and your radio talks, has the American government itself been against you? *A.*—Well, I wouldn't know how to answer that question.

Q.—Well, isn't—don't you claim that the American government itself—well, I will just say here on page 276 of your book isn't it a fact you state, "The American Government itself now came out openly against" him? *A.*—I think I was referring to the State Department, yes, sir.

Q.—The State Department of the United States government? *A.*—(No answer.)

Q.—Now, it says in the article, you see this first paragraph, it says that you are a quack and it said about making money. Then we said in the article that the various governmental departments—that you kept on making money, notwithstanding the efforts of the various governmental departments and agencies. Then down here you also state that you have been damaged because the article says that when your radio station was removed from Kansas—don't you say from Kansas—"he departed to Mexico, obtaining a license from the Mexican Government." Now, when you lost your license to broadcast in Kansas, Doctor, it is a fact that you then went to Mexico and made arrangements for the station across the river from Del Rio, isn't it?

Mr. Morriss Sr.:—Same objection.

THE COURT:—Overrule the objection.

A.—Yes, sir.

Q.—And you are now broadcasting from station XERA at Villa Acuna, Mexico? *A.*—Yes, sir.

Q.—Just across from Del Rio, Texas? *A.*—Yes, sir.

Q.—That is true, isn't it? *A.*—Yes, sir.

Q.—Isn't it a fact that "all sorts of efforts have been made to remove him from the air, but apparently without success," that is true? *A.*—All sorts of efforts were made to remove me.

Q.—Here is one thing Dr. Fishbein said in the article which you now complain of. He said "all sorts of efforts have been made to remove him from the air, but apparently without success"? *A.*—Yes, I quite agree with that part of the article. All sorts of efforts have been made to try to close me down, put me out of business. I agree with them.

Q.—So that is true, isn't it? *A.*—I agree with Dr. Fishbein on that.

Q.—Now, the article says—

Mr. Morriss Jr.:—Just a minute. In connection with that we would like to say "all sorts of efforts," that would necessarily mean by some particular person or individual. The question would be on whose part. If all sorts of efforts have been made by the American Medical Association, by Dr. Fishbein, or at his instigation, why of course then it would perhaps be of some bearing, but if somebody else, some wholly independent concern, we just think that that sort of thing, that that kind of statement is a shotgun statement and doesn't show anything about who made all sorts of efforts or at whose instigation, and therefore would have no place in this evidence.

THE COURT:—You seem to be in disagreement with your client. He says he is heartily in agreement with it.

Mr Morriss Sr.:—I understand, but we are talking about the legal aspect, not talking about whether they were made, but it is not shown by whom.

THE COURT:—I don't know that I understand what the nature of your objection is, what is before the court, but if you are objecting to that last question that was asked and the answer, I will overrule your objection.

Q.—Now, in connection with the sentence in the article that you complain of is right now, that is, February 1938, "right now he is not doing much in the way of goat gland transplanting." That is correct, isn't it, Doctor? You weren't doing any at that time, were you? *A.*—I haven't been doing any transplanting since 1933.

Q.—So that sentence is correct, "right now he is not doing much in the way of goat gland transplanting" Is that correct?

THE COURT:—He answered the question, counsel. Pass on. He answered it.

Q.—Now, then, we come to this part here where it says "he reads," the article says, "he reads the symptoms of patients and then by remote control prescribes the pill that they should use." Well, isn't it a fact that people write in to you, ever since you have been broadcasting, they write in to you telling you of their ailments and symptoms and so forth? *A.*—Yes, sir, they write to me but I don't go back over the radio and answer their letters and prescribe for them.

Q.—Don't you tell them over the radio what prescription to take? *A.*—No, sir.

Q.—Have you ever done that? *A.*—Not since 1930.

Q.—Not since 1930? *A.*—No sir.

Q.—Well, how do you then—do you have any prescriptions now that are numbered? *A.*—No, sir.

Q.—When did you quit numbering your prescriptions? *A.*—1930.

Q.—That is the last time? *A.*—Yes, sir.

Q.—Do you sell 1022 now? *A.*—No, I don't sell it.

Q.—Do you— *A.*—Unless you can call selling—you come into my hospital and become a patient and I use it on you. I sell it that way, but I don't sell it out through the mail or advertise it to mail to somebody, you send me so much money and I will send you 1020. I don't do that.

Q.—When did you quit? Say the folks would write in to you and say "I hurt so and so and my symptoms are so and so," and you would take the letter and read it over the radio on broadcasts and say, "I advise this lady to take prescription No. 50." *A.*—I quit that in 1930.

Q.—In 1930? *A.*—Yes, sir. I promised the Federal Radio Commission—we had the hearing in June 1930—that I wouldn't read those letters and prescribe any more over the radio, and I haven't since.

Q.—But how many years before 1930 did you do that? *A.*—I only did it for about a period of six months. I began it in the fall of 1929, and the whole thing was over in June 1930.

Mr. Morriss Jr.:—Now, then, we move the exclusion of that.

THE COURT:—Overrule the motion.

Q.—Did you ever advertise in any of your literature or say over the radio if the people would send a dollar or two dollars in that you would advise them what was the matter with them and tell them what medicine to take? *A.*—No. I said if anybody wanted a personal letter from me to send two dollars and I would write them a personal letter of advice. I didn't say I would send anything. I had to do that to overcome so many requests. There were thousands of requests wanting me to write them something, and it was utterly impossible to do it.

Q.—Now, then, here is another line you complain of: "In addition, of course, he has a hospital in Del Rio where he performs some peculiar operations." During that time back there before 1930, when they would write the letters and then you would prescribe over the radio, didn't you have an arrangement with the druggists throughout Kansas where they would have this medicine in the drug stores numbered Dr. Brinkley prescription No. 50? *A.*—The druggist had the prescriptions. I had the druggists in five states, which we would call the primary service area of the radio station. In other words, our trade territory. And these prescriptions were sent to the druggists for their files, so if you came in and said No. 50 the druggist would mix it and compound it according to that prescription. That is the way it was handled.

Q.—How many of those numbered prescriptions did you have, do you remember, about? *A.*—It seems to me like they ran somewhere from one to fifty, something like that.

Q.—And the people would write in and then you would read the letter over the radio? *A.*—Yes, sir.

Q.—And you would say "I advise this lady and people that are suffering like her to go to the drug store so and so," whatever drug store she lived by, "and get prescription number so and so"? *A.*—No. 50 or 60 or 70, whatever it was, yes, sir.

Q.—And, of course, you got some commission or divided the profits with the druggist, didn't you, Doctor? *A.*—I didn't get a commission. I had an understanding with the druggists that they were to send so much of their total weekly sales to be applied against the weekly broadcasts. In other words, if that wasn't done he would be getting it for nothing.

Q.—In other words, you made some money? *A.*—I didn't make any myself, there was no intention to make money out of it. It went to the station for operating expenses.

Q.—Now, the article said you operated a hospital at Del Rio. That was correct, wasn't it? *A.*—Yes, sir.

Q.—Now, it said "where he performs some peculiar operations." Well, the operation that you performed on the enlarged prostate, that was different from the standard operation performed for that by the regular doctors, wasn't it, Doctor? *A.*—Well, the operations I perform are different from the rank and file performed by the regular doctors. Of course, I do transurethral work—

Q.—I just wanted to cover that point. You performed operations here in Del Rio that were different from the regular standard practice, as you understand it? *A.*—Yes, sir.

Q.—That was adopted by most of the physicians? *A.*—Yes, sir.

Q.—That is true, isn't it? *A.*—Yes, sir.

Q.—And the main one of these is what you call the compound prostate operation for elderly gentlemen, that was the main one, wasn't it? *A.*—I call it the surgical technic. It used to be called the compound operation.

Q.—And in which it says "He merely injects a little mercurochrome." You did in that operation, in many cases, inject some mercurochrome into the tubes passing from the main sex glands, didn't you?

Mr. Morriss Sr.:—Just a moment, counsel. He misconstrues the statement the way he words that.

THE COURT:—That is a matter of argument. You will have an opportunity to examine the witness.

Q.—In some cases, of course, you would do more than inject the mercurochrome, is that right? *A.*—In all cases I do more.

Q.—You do more? *A.*—Yes.

Q.—All right. Now, you don't collect from the patients yourself, do you, Doctor? *A.*—No, sir.

Q.—You have some manager, business manager, that makes arrangements about getting paid for the work? *A.*—That is right.

Q.—And do you usually inquire, ordinarily and usually do you inquire of your business manager to see whether your people can pay or will pay before the work is done? *A.*—Yes, sir.

Q.—Ordinarily? *A.*—No, sir.

Q.—Most of the time you do the work without knowing whether you are going to get the money? *A.*—I thought you meant did we make inquiry to find out whether the patient had money or not.

Q.—No, sir. Here a man comes, he wants to be operated on and you think he is going to be operated on, whether you endeavor, as a business man of course you endeavor to see if you are going to get your money if you operate on that man? *A.*—That is right, yes, sir.

Q.—Now, in reference to these—you get a whole lot of letters, don't you? *A.*—Quite a few, yes, sir.

Q.—And you answer—no, I believe you state now that you don't answer those letters over the radio? *A.*—No, sir.

Q.—But you develop a lot of your business through the mail by correspondence and letters you get from people and letters you write to them? *A.*—Yes, sir.

Q.—Now, since you have been in Del Rio you haven't used the goat gland operation, as I understand it? *A.*—None whatever, no, sir.

THE COURT:—He has been asked about that several times, counsel. Don't ask him about that again.

Mr. Brown:—I beg your pardon.

Q.—You have been using what you call your new surgical technic. What did you say awhile ago it was? *A.*—Surgical technic, yes, sir.

Q.—Now, what has been the result in operating on the prostate glands by the use of this new technic? Has it reduced the enlarged glands? *A.*—Yes, sir, it does.

Q.—Isn't it a fact that you claim that it clears the infection out of the entire human body? *A.*—Not the operation itself, but by the ministration of drugs and removal of focal points of infection—

Q.—Excuse me— *A.*—Let me explain something to you so you can ask me better questions. When we speak of our surgical technic at $750, that not only refers to a man's prostate gland but refers to dental work and everything.

Q.—I understand about that. *A.*—We take a case to clean him up and we call that our surgical technic, guaranteed service for lifetime. That is what we are talking about, complete service for the patient for whatever he needs.

Q.—This book published in 1937, on page 301, don't you claim it greatly increases the sexual powers of men and women? *A.*—Well, the work we do—

Q.—No, excuse me. Didn't the book you published in 1937 say that? *A.*—Probably it did.

Q.—Overcome depressed mental conditions, that is what you said in the book, wasn't it? *A.*—I think so, yes, sir.

Q.—Didn't you say in the book "He has, as a matter of cold sober fact, changed the color of his patient's hair, smoothed the wrinkles out of their faces, and turned their complexions from a sickly pallor of old age and disease back to the ruddy glow of health"? *A.*—That is true.

Q.—Is that true? *A.*—Yes, sir.

Q.—What is the closest to $55,000 a week that you ever made? This thing says here it has been reported that sometimes you have made $55,000 a week. What is the closest to that you ever got, that you recollect? *A.*—I don't think that I ever exceeded over $120,000 to $125,000 in a month.

Q.—In one month? *A.*—So if we divided that by four it wouldn't be that much.

Q.—You don't think that Dr. Fishbein ought to pay you any damages because he said you made $55,000, do you?

Mr. Morriss Sr.:—Just a moment, that is argumentative.

Mr. Brown:—I beg your pardon, your honor.

Q.—Doctor, you have been the possessor of three fine yachts, haven't you? *A.*—Well, I am happy—

Q.—I say you have been the possessor of three fine yachts, haven't you? *A.*—I own three yachts, yes, sir.

Q.—Well, how many men does it take to run the one you have now? *A.*—It takes twenty-one men.

Q.—A seagoing yacht? *A.*—Yes, sir.

Q.—You don't want to say it is a fine yacht, do you? *A.*—The one I have now is a mighty nice yacht, yes, sir.

Q.—Thank you. Now, when you go and come across the seas on your summer trips you go in the finest boats, don't you? *A.*—I try to go on good boats, seaworthy boats.

Q.—Are there any better boats than the *Queen Mary* or *Normandie?* *A.*—If there were I would have been on them.

Q.—You go on the finest boats and stop in the best suite on the boats, don't you? *A.*—I try to, yes, sir.

Q.—Isn't it a fact that for yourself and your wife and tutor and little boy you paid $1,900 to get from France back to the United States on the *Normandie?*

Mr. Morriss Jr.:—We object to that as being irrelevant and immaterial.

THE COURT:—Overrule the objection.

Mr. Morriss Jr.:—Note the exception.

A.—I don't know what I paid the American Express Company. I have an unlimited charge account with them, and after the trip was over, five or six months later, they sent a statement in to me and my bookkeeper paid the bill.

Q.—You don't know whether it was $2,000 or $5,000, do you? *A.*—No. I suppose it was eighteen hundred or two thousand dollars, I suppose, something like that.

Q.—Well, in traveling across there on the boats do you and Mrs. Brinkley talk to the passengers or not? *A.*—Oh, some of them, if we meet them and get acquainted with them, yes, sir.

Q.—Doctor, the money is still rolling in to you, isn't it, and you make most of your money out of the practice of medicine, don't you? *A.*—Yes, my main income is from my hospital work.

Q.—And the money is still rolling in, isn't it? *A.*—Well, I don't consider it that way.

Q.—I don't mean rolling in. This thing here says rolling. In other words, you are still making good money as a physician, aren't you? *A.*—I have a nice practice.

Q.—Now, when you were here, when you were here in Del Rio you didn't have a separate hospital, did you? Was your hospital in the Roswell Hotel? *A.*—That is right, we used three floors in the Roswell for hospital purposes.

Q.—Now, how many rooms does that downtown hospital have, the downtown hospital you have in Little Rock? *A.*—Oh, about fifty.

Q.—How many patients will the downtown hospital accommodate? A.—We can squeeze in forty.

Q.—Yes, sir. Was that a two story building or a three? *A.*—Two stories.

Q.—Was that an old building or was it constructed as a hospital? *A.*—That was St. Luke's Hospital, owned by one of the surgeons down there that died, and I bought it afterwards.

Q.—And then you have another hospital out in the country about sixteen miles? *A.*—Yes. We call that the Country Club Hospital.

Q.—That is an old country club out there? *A.*—No, it was the Shriners' Country Club out there.

Q.—How many rooms has that got, about? *A.*—You want to know how many patients I can keep out there?

Q.—Yes, sir. *A.*—So I must tell you we can take care of about fifty patients out there. It is in a different arrangement from the rooms at the other place. There is a large receiving, reception halls, and dining rooms and dance rooms.

Q.—Do you own that? *A.*—Yes, sir.

Q.—How many acres are there there? *A.*—Three hundred and seventy-five.

Q.—It was at one time used as a country club? *A.*—I want to correct that statement. You asked me if I owned it. I don't know whether it means anything or not, but at least I want to try to tell the truth. That is in a trust estate for my son, and I am the trustee and manage the trust estate. Whether that makes any difference in the answer I don't know.

Q.—Now, those three floors that you say you had, did you have the whole of the three floors in the Roswell Hotel here in Del Rio? *A.*—Well, we had all of the mezzanine floor, and if I am not mistaken I think—I think we had two floors above that, completely to our own use.

Q.—How many rooms were in there altogether? *A.*—I don't remember. We used the mezzanine for administrative purposes and operating room. We had our sterilizing equipment and laboratory equipment in the basement.

Q.—Were any patients on the mezzanine floor? *A.*—No, they were all examining offices and diagnostic offices and treatment offices and operating room and business offices and things like that.

Q.—Dr. Brinkley, under this new technic I understood from Mr. Petermeyer that testified yesterday, that you cut the ducts between the testicle and the epididymis, or the fold, or whatever you call it? *A.*—No, counsel, we don't cut, we ligate.

Q.—You tie it? *A.*—Yes, a linen suture.

Q.—It is just the same as if you cut it, nothing can come through there? *A.*—As long as the suture is on there the patient would be sterile. Take the suture off and he is fertile again.

Q.—You say about fifteen of those little ducts from the testicles to the epididymis, each testicle has about fifteen. Now, when you perform the operation do you tie all the little ducts between the testicle and the epididymis? *A.*—In some cases they are all tied off. In other cases only a part of them.

Q.—Well, didn't you say in your deposition that that—what was the name of that case, Parnell case; when they took your deposition didn't you say three different times you cut them all, you cut all of the efferent duct, tied them off, excuse me, ligated all of the efferent duct reaching from the testicle to the epididymis? *A.*—If my memory hasn't gone bad on that deposition, I stated we ligated a part of that, not all of them, and I think the counsel asked me "Well, how many?" and I said "Probably ten, eight or ten or eleven," something like that.

Q.—Isn't it a fact in the deposition you said three different times you ligated all of them? *A.*—We do ligate all of them in certain cases, yes, sir.

Q.—I can show you that, as to how it came up, as to that time. Now, isn't it a fact that in that deposition when they asked you about it you said "Question: Now, what do you mean by ligating those ducts, state that technique to us, please. Answer: Well, it is talking of the fifteen efferent ducts on each side, approximately that number, tying off some of the sections of the testicle so secretions can't pass through the ducts to the epididymis. I think that is about as plain as I can say what I mean by ligate. Question: Do you mean tie off with catgut or linen? Answer: Yes, sir, we use linen. And you sometimes tie of all fifteen of the efferent ducts. Question: Of both testicles? Answer: That is right. If you don't do it on both sides you might as well not do it at all. Question: Well, do you do it on both? Answer: Yes, sir. Question: All fifteen ducts? Answer: Yes, sir. Question: Do you ligate the vas as well, Doctor? Answer: No, sir. Question: That is the main tube that goes on up, isn't it"—excuse me just a minute. *A.*—I think I had already answered you.

Q.—I want to show how you answered in the deposition. *A.*—Yes.

Q.—Doctor, last Sunday night isn't it a fact you offered hundred dollar prizes over the radio to folks that would write the best letter on good health and send it to you and the names of two people that were sick and two people that had enough money to pay their way to Arkansas? *A.*—I didn't specify the number.

Mr. Morriss Jr.:—Just a moment. We desire to make an objection that that isn't a relevant time, that it is not a matter at the time that is in issue.

THE COURT:—Don't the courts hold these damages, if there are any damages, are not only retroactive but perspective?

Mr. Morriss Jr.:—Yes, sir.

THE COURT:—The extent of the injury not only relates to the present and past but is carried forward to the future.

Mr. Morris Jr.:—Yes, sir.

THE COURT:—Accordingly, it would be proper to inquire as to the present condition, what he is doing, how his business is, and that sort of thing.

Mr. Morris Jr.:—We don't think it is proper to go into any matters now that wouldn't reflect upon, wouldn't shed any light on the issue of the damages at all.

THE COURT:—Well, the general allegation is made as to the lack of ethics and the fact that he is a quack. I think what he is doing now would be relevant. I will overrule the objection.

Mr. Morriss Jr.:—Note the exception.

Q.—That is a fact, isn't it, Doctor? On last Sunday night, substantially, you said you wanted to make a hundred dollar prize, to give it to the one that writes the best letter on health and sends to me the name of two sick people who are able or have money enough to pay their way to Little Rock, Arkansas? *A.*—No, sir, that isn't the way I said it.

Q.—All right, how did you say it? *A.*—I said "Next Wednesday we are going to divide one hundred dollars in cash into ten equal prizes."

Q.—Oh, yes. *A.*—What I want you to do is to take one sheet of paper and complete this in twenty words or less: I consider good health my most valuable possession because—because what? Complete that in twenty words or less; on a separate sheet of paper send me the names and addresses of men that you are personally acquainted with, that you personally know to be sick and in need of our services, physically and financially able to come to see us.

Q.—Physically and financially able to come to see you where? *A.*—Little Rock, Arkansas.

Q.—Yes, sir. And last night isn't this a part of your radio address, last night, "Do you want to participate in the distribution of $500 cash without your having to send any bottle cap or carton or to buy anything, just your intelligence? You are thinking we will listen on the air first. This opportunity will close to you, but between now and April 30th there are going to be many men and women receiving distributions." You said

that last night, didn't you, Doctor? *A.*—I never heard the advertisement. I don't know a thing about it. All I can answer you about it is I told the announcer to make a contest along similar lines and $500 would be awarded May 5th.

Q.—Who was doing that talking, you or— *A.*—I wasn't over at the station, I wasn't over there. I don't know, I guess Mr. Don Howard, the announcer, I suppose.

Q.—They didn't use a record of your voice or anything of that kind? *A.*—He may have. Maybe I made a record before I left and went back to Little Rock. I made a record last—I made a record last Monday morning, and I think I put on that record—

Q.—This $500 business? *A.*—I believe I did.

Q.—All right. And that came over the air last night, didn't it? *A.*—I don't know, I never listened to the station. If you heard it I guess it did.

Q.—Then you don't—excuse me. "First prize $100 in cash; second prize $50; third prize $25; fourth prize $10 in cash; five prizes of $5 each, and then 290 other cash awards of $1 each; 299 cash awards. Each entry you send in has 299 chances to win as much as $100 in cash, and listen, folks, it is easy. All you have to do is think, just think a little and be real sincere and real honest. Here is the slogan, 'I consider Dr. Brinkley the world's foremost prostate specialist because,'" and that is the blank? *A.*—Yes.

Q.—And complete that in twenty words or less on one sheet. That is the idea, isn't it? *A.*—Yes.

Q.—That is practicing medicine, isn't it? *A.*—What?

Q.—Practicing surgery, is that practicing medicine, practicing surgery, please? *A.*—No.

Q.—All right, sir, "and complete that in twenty words or less on one sheet. On the second sheet I want you to be real sincere and know what you are doing. Just mail at least five names and addresses of men that you are personally acquainted with. Be sure you know them, be sure you know they are sick, and be sure you know that they are in need of Brinkley's hospital service. Be sure you know, be sure you know that they are physically and financially able to come to see us. Just those five names and addresses on one sheet of paper. You complete the slogan 'I consider Dr. Brinkley the world's foremost prostate specialist because'—blank—on a second sheet of paper, and send it to Dr. J. R. Brinkley, Little Rock, Arkansas, and this prize ends April 30th and this cash will be distributed May 5th. Now, folks, for your own health's sake, for your wife's sake, for the sake of your loved ones, if sick don't neglect to send for the book. Don't sit down there and listen to me and do nothing about it. If undecided, don't know what to do, then listen, then listen to what you can do. Come and see us. I don't mean come see me or come to see the doctors and the nurses, come to see my patients. We have two hospitals full of people. Come in the front door or even in the back. Meet the patients. See what is happening to them. They are just people like you. And then decide what you want to do." Now, that was your advertisement that went out over the radio last night, wasn't it? *A.*—I don't know whether it went out last night or not, but that is my advertisement, so what difference does it make?

Q.—Probably this week? *A.*—Yes, sir.

Q.—And you send it out over this powerful radio station right across the river, isn't it? Is that the station it goes over now? *A.*—Yes.

Q.—How far will that reach, to Canada and all over the United States? *A.*—It doesn't get into Canada because of a 50,000 watt station at Toronto knocks it out.

Q.—Practically all over the United States, is that right? *A.*—Not in the East. The primary service area of the station is a line drawn through Louisiana, Alabama, Georgia, up around through the Carolinas and Virginia, and skirting Pennsylvania, a part of Ohio and across along the circular line passing through the Great Lakes and on down to the coast. Of course, now, of a morning when we broadcast the Canada station is not on and the station will reach out all over the United States and Canada, but I suppose you are talking about evening broadcasts.

Q.—Doctor, is this kind of advertising, is it usual for practitioners of medicine in the United States to conduct prize contests such as this? *A.*—Not if they want to stay in the American Medical Association.

Q.—Excuse me. Is it usual for practitioners of medicine in the United States to conduct these prize contests? *A.*—No, sir, I don't think so.

Q.—Do you know of anybody else that is talking over big radios offering cash money? *A.*—No, sir.

Q.—Do you know of anybody sending out certificates with red seals on them—give me that—with red seals on them, telling them, if you will come down to the hospital and write the best piece of any ten people we will put a seal on it like a notary seal, tell them if they will do that, we will give them an automobile? *A.*—No, I don't know of anybody doing that.

Q.—You are the only one doing that? *A.*—As far as I know.

Q.—Isn't it a fact that on March 22, this year, that you sent out this broadcast over your station: "What is the use of trying to do anything when there is cancer in your prostate gland, because leaving that infection in there for years—there must have been something left out there—what are you going to do about it? You take a risk. Why take a chance? You are sick and miserable. Is your property doing you any good? Is that money doing you any good? Yet you have worked and made money, made property for the purpose of using it when you need it, and when a man and woman is sick is there any time in your life when you need to spend that money more than a time to get your health back? If you don't supply the money you are going to die. You are not going to know health until your body becomes so diseased and so great a sum of money that you can't mortgage the property"—that must be left out— *A.*—It is garbled.

Q.—Yes, I withdraw it. "That is the picture that some men and women—of some men and women who neglect their lives, poor houses at the end of the road, and it might not have been, might not ever have been true if you had acted promptly. That is, don't continue to take treatments that are doing you no good. I believe that by the time of the end of this broadcast comes you are going to go and call or mail reservations to come to Brinkley's hospital in Little Rock as thousands of people have done and start life again down that road on the poor house. This is Dr. Brinkley urging you to do it now." There are some portions left out there. *A.*—It is garbled.

Q.—That is substantially it? *A.*—Some of it is so and some don't make sense.

Q.—Yes, I agree. Did you ever say over the radio, Dr. Brinkley, in effect, "Come to me and don't let your family doctor two dollar you to death"? *A.*—Yes, sir.

Q.—How many times have you said that? *A.*—I don't know how many times.

Q.—Throughout how many years have you said that? *A.*—I only started that this year. That is a new one.

Q.—Is it? How many times have you said it in the last year? That is, "Come to me, don't let the old family doctor two dollar you to death." *A.*—I didn't say the old family doctor, I said don't let your doctor two dollar you to death. I don't think I said old family doctor.

Q.—You didn't want to say that, did you? *A.*—Oh, I wouldn't say that.

Q.—Oh. Doctor, now, where do you claim, please, that you got your medical education? *A.*—I had three years in Bennett Medical College; one year in Eclectic Medical College in Kansas City, Missouri.

Q.—When did you go to Bennett? Was that in Chicago? *A.*—Yes, sir. I matriculated in 1908 and left there when the junior year was over, about the 1st of June 1911.

Q.—Did you go to one right after the other? I mean no skipping there, three solid years? *A.*—1908 to 1909, the freshman year solid; 1909 to 1910, sophomore year, was solid, and from the fall of 1910 to 1911 I missed about six weeks of the first part of junior year and didn't get back into school until some time, I think, around the early part of November.

Q.—Now, where were you during those six weeks that you missed? *A.*—Telegraphing in New York.

Q.—For whom were you working? *A.*—Western Union.

Q.—Do you remember what months those were? *A.*—I went down to New York—I think I went down from Montreal, I was telegraphing in Montreal, and I left Montreal, I think it was around September, and went down to New York and resumed work for the Western Union there until some time in November.

Q.—About how long had you been in Montreal? *A.*—I went to Montreal when I got through with my sophomore year in the summer of 1910, and I spent the summer up there.

Q.—And then you missed, you say, some of the fall term, about six weeks? *A.*—Yes, sir.

Q.—And then you completed your junior year at Bennett College at the end of the spring term? *A.*—Yes, sir, in 1911.

Q.—And when did you go to this Kansas City Eclectic College? *A.*—1914 to 1915.

Q.—So you skipped how many years in there between the Bennett College and the Eclectic College? *A.*—Well, out of school in the summer of 1911 and not back in school until the fall of 1914. That was three years.

Q.—And during that time what were you doing? *A.*—During the summer of 1911 and fall of 1911 I was practicing medicine in western North Carolina, where I was born and raised, by permit of the secretary of the medical board, who knew I was an undergraduate. Practiced there until some time, I think it was in October of 1911, going down to Jacksonville, Florida, for a couple of weeks, where I worked for the Western Union Telegraph Company, and left there and went to Whiteville, North Carolina.

Q.—When was that, do you remember? *A.*—Well, I think I went to Whiteville, I think I landed in Whiteville right close to the end of 1911. I can't be sure.

Q.—Yes, sir. *A.*—And that has been so long ago, twenty, nearly thirty years ago. I am trying to give you approximately.

Q.—All right, sir. *A.*—And some time in there some place I left Whiteville, I don't remember when, I can't remember when I was there, and I went up to St. Louis, Missouri, and stayed around there for a short time and came on back to Tennessee, went to Danrich, Tennessee, practiced medicine at Danrich during the summer of 1912, the summer of 1912 I was practicing medicine at Danrich, Tennessee. During the fall of 1912 I moved down to Knoxville, Tennessee, and I practiced around there somewheres toward the close of 1912 and the beginning of 1913, some time during the beginning of 1913—

Q.—Now, you didn't have a regular license at that time, as I understand it? *A.*—I had a license, undergraduate's license.

Q.—All right. *A.*—I had an undergraduate's license in Tennessee. I was practicing legally.

Q.—Did you ever work and practice down at Greenville? *A.*—I opened an office there for another man. I never practiced there, never have practiced there.

Q.—What was the man's name? *A.*—Mr. Burke.

Q.—What was your partner's name in that venture? *A.*—His name was Crawford.

Q.—Isn't Burke and Crawford the same man? *A.*—No, Mr. Burke was the owner of a chain of medical offices over the country.

Q.—What kind of diseases did that chain treat? *A.*—Treated two, gonorrhea and syphilis.

Q.—Now, when you were there at Greenville, that institution you opened up at Greenville, they had paid ads in the paper advertising they would treat gonorrhea and syphilis, didn't they? *A.*—I can't remember.

Q.—What was the name of the institution, the branch office you opened up at Greenville? *A.*—The Electro-Medic Specialists, I think that is the name of it.

Q.—Do you remember whether—do you remember whether you did any advertising there? *A.*—Oh, yes, there was many newspaper advertisements run.

Q.—Electro-Medical Doctors? *A.*—It might have been doctors.

Q.—Where was the store located? *A.*—You mean the office?

Mr. Morriss Jr.:—Just a minute. It seems to indicate that this was not advertising Dr. Brinkley, he was not—this was advertising of somebody else, where he simply went to open the place of business for him, so it isn't a question of what the advertisements say for the other doctor, or some other doctor. We think that that wouldn't have any bearing here, and besides it is far too remote.

THE COURT:—Overrule the objection.

Mr. Morriss Jr.:—Note the exception.

Q.—Well, it is a fact that you went down there to Greenville even while just a student doctor, before you ever got your diploma you went down there to Greenville working for a man that had a chain. Was that fellow Burke a doctor? *A.*—No, sir.

Q.—And he had these offices in different towns? *A.*—Yes, sir.

Q.—And they treated gonorrhea and syphilis? *A.*—That is right.

Q.—You went down there and opened up to start the office for him in Greenville, North Carolina? *A.*—South Carolina.

Q.—And when you opened up and got ready to start, isn't it a fact you put big ads in the newspaper that you were opening up this office for the treatment of gonorrhea and syphilis? *A.*—I know advertisements were run in the paper announcing the opening of the office. That is what my business was, to advertise the opening of the office.

Mr. Morriss Sr.:—In connection with that we want to make certain objections. I guess your honor wants to quit now.

THE COURT:—You have made your objection, and it was ruled on.

Mr. Morriss Sr.:—There is further matter coming on. We will try to find out just what it is.

THE COURT:—We will take a recess.

At this time a recess was taken until 2 p. m. of the same day, at which time the following proceedings were had, in the presence of the jury:

Questions by Mr. Brown:

Q.—Doctor, when we closed just before dinnertime you had started to say—you had stated that you went down to Greenville and opened up a branch there for Mr. Burke, and that office was going to treat gonorrhea disease and syphilis, and then the question was asked you and I don't think it was answered, whether you had put ads in the paper when you opened up that office there advertising the office to treat gonorrhea and syphilis. *A.*—I can't remember the contents of the ads, but I know there was many ads running in the newspapers announcing the opening of the office.

Q.—And did you practice there in connection with that? *A.*—No, sir.

Q.—Now, what kind of a degree or diploma did you get when you say you got three years? *A.*—The Bennett College in Chicago and one year at the Eclectic College.

Q.—And where was that? *A.*—At Kansas City, Missouri.

Q.—And what kind of a degree or diploma did you get from the Eclectic College at Kansas City? *A.*—A degree of M. D., Doctor of Medicine.

Q.—Doctor of Medicine? *A.*—Yes, sir.

Q.—Did it state on there whether it was an Eclectic College, or not, or an eclectic degree? *A.*—That was the name of the university, the Eclectic Medical University, that was the name of the school.

Q.—What does that mean, eclectic? *A.*—It is a word meaning to select the best, I believe that is the literal translation of the word, select the best.

Q.—Isn't it a fact that Arkansas is the only state in the Union now that will issue a license to practice medicine when a man has gotten a diploma from an eclectic college? *A.*—I don't know.

Q.—Do you know of any other state besides Arkansas that will let you practice if you have graduated from an eclectic school? *A.*—Oh, yes; I think you can go before many medical boards and get a license to practice.

Q.—Just upon your degree or diploma? *A.*—You would have to take the state board examination.

Q.—Have to take the examination? *A.*—Yes, sir.

Q.—Now, when you graduated from the Eclectic College of Kansas City it is a fact, isn't it, that the whole class went at one time down to appear before the Eclectic Board in Arkansas? *A.*—I know most of us went down, but I don't know whether all went down or not; I don't think all went.

Q.—Practically all? *A.*—The majority of them did.

Q.—Did one of the professors go along with them? *A.*—Yes, sir.

Q.—And there they were admitted to practice medicine in the state of Arkansas? *A.*—They passed the examination, yes, sir.

Q.—That examination was before what is known as an Eclectic Board? *A.*—Eclectic Medical Examination Board, yes, sir.

Q.—Now, in reference to your licenses to practice medicine, I don't think you answered the question as to whether you had a temporary license to practice medicine in California? *A.*—Yes, sir.

Q.—And then you applied for a permanent license to practice medicine in California, didn't you? *A.*—Yes, sir.

Q.—Did you make an application to practice—that was refused? *A.*—Yes, sir.

Q.—Did you make an application to practice medicine in the state of Illinois? *A.*—Yes, sir.

Q.—Was that refused? A.—Yes, sir.

Q.—As late as 1938 isn't it a fact that you again applied to the state of Connecticut to practice in Connecticut? *A.*—In 1938?

Q.—Yes, sir. *A.*—No, sir.

Q.—Within any recent years have you reapplied for a license to practice in Connecticut? *A.*—No, sir.

Q.—My information may be incorrect. *A.*—You are wrong there.

Q.—Since your license was revoked in Connecticut you never have reapplied? *A.*—No, sir.

Q.—Lately have you reapplied to any other state to practice medicine? *A.*—No, sir.

Q.—When were you first married, please, Doctor? *A.*—1907.

Mr. Morriss Sr.:—We just say that anything of that kind is incompetent, irrelevant and immaterial to any issue in this case, to ask when he was first married, and we can see no pertinency of the inquiry here, and we shall object to all testimony along that line.

THE COURT:—I think a general background from the witness and the plaintiff would be admissible.

Mr. Morriss Sr.:—We think it would not be admissible on any ground, and it is a matter that can't be legitimately used for any purpose; and the only purpose for which it could be used would be an improper one, and, therefore, we shall object to it and all testimony pertaining to it.

THE COURT:—Reference is made to the family in the article that the suit is on. I will overrule the objection.

Mr. Morriss Sr.:—Note our exception.

Q.—When were you first married, please? *A.*—1907.

Q.—And who did you marry? *A.*—Sally White.

Q.—Now, when did you marry your present wife? *A.*—1913.

Q.—And what was her name, please? *A.*—Minnie Jones, Minnie T. Jones.

Q.—That was in 1913? *A.*—Yes, sir.

Q.—And did you get a divorce from your first wife? *A.*—Yes, sir, I did.

Mr. Morriss Jr.:—We make the same objection to all of that, if your honor please.

Q.—When was that? *A.*—In the early part of 1913.

Q.—Was that a citation by publication?

THE COURT:—Counsel, I don't think there is anything gained in trying out the divorce case.

Mr. Brown:—I am not going to do it, your honor.

THE COURT:—It is not in issue here. I think it is all right to show the constituents of his family and the effect the article might have had on him, but any questions as to the rights or wrongs of the divorce case—I don't know what they are, but I am not going to inquire into that.

Q.—Did you remarry your present wife? *A.*—I did.

Q.—In what year, please? *A.*—In 1915 or 1916, I forget which.

Q.—Now, your first wife, did she get a divorce from you, or did you get a divorce from her—you say she got one from you too?

Mr. Morriss Sr.:—That is objected to.

THE COURT:—What is the materiality of that?

Mr. Brown:—Just to show the whole picture of it, your honor.

THE COURT:—Yes, I know, but the picture may be too broad, and take too long and I don't see that we are interested in that; that is not an issue in this article; and outside of showing what the constituents of the family are and who they are, why, I don't think it is material to go into the question of the divorces.

Mr. Brown:—We except to the ruling of the court.

Q.—Now, when you were down there in Greenville and opened up that office there, weren't you charged with forgery and practicing medicine without a license? *A.*—No, sir.

Q.—Weren't you arrested there for something?

Mr. Morriss Sr.:—We object to that for all of the reasons. That is going back to a period, we assume from the inquiry, to 1911.

Mr. Brown:—1913.

Mr. Morriss Sr.:—Well, 1911 to 1915, and the Doctor said he had been practicing with irregularity since 1915, and we say it is immaterial and irrelevant and too remote and that it is not the best evidence and that it is of a nature calculated to prejudice his rights without any rule of law which would admit it, and it is not a proper matter of impeachment.

THE COURT:—I will overrule the objection.

Mr. Morriss Sr.:—Note our exception.

A.—No, sir, I wasn't arrested there. I was arrested in Knoxville, Tennessee, on a charge that was brought down there.

Q.—What were those charges, please? *A.*—I don't know what the charge was, but I know that this fellow, Crawford—

Q.—Excuse me, but I understand about that. Isn't it a fact that down at Greenville they charged you with forgery and practicing medicine without a license? *A.*—Not to my knowledge; I don't even know what the charge was.

Q.—Did they take you back to Greenville? *A.*—I voluntarily went back.

Q.—Were you in jail there for any time? *A.*—No, sir, I didn't stay in jail while I was at Greenville, I stayed—

Q.—At the sheriff's house, or something? *A.*—Yes, sir, at the sheriff's house about forty-eight hours, and I paid off these bad checks that this fellow had written.

Q.—And your lawyer's name was Burke? *A.*—I don't remember.

Q.—And the man with you, he was named J. E. Crawford? *A.*—Yes, sir.

Q.—And did your father put up the money for these checks, or your father-in-law?

Mr. Morriss Sr.:—We object to that.

THE COURT:—I think that is immaterial.

Mr. Brown:—I just had this in mind, it is shown that he is charged with forgery and we just want to show that his family rustled up the money and paid the fellow that had the checks and they never did prosecute the checks, and that is as to why it wasn't concluded with a conviction or otherwise, and that is what we had in mind.

Mr. Morriss Sr.:—We will ask the court to instruct the jury not to consider the improper remarks of counsel.

Mr. Brown:—I just wanted to tell the court what I had in mind.

THE COURT:—Yes, sir, but you must not in telling the court attempt to testify yourself until the court has an opportunity to rule on those matters. I will sustain the objection to the question as asked, and I will instruct the jury to disregard the remarks of counsel, as they are not competent.

Mr. Brown:—We except to the ruling of the court.

Q.—Now, when you were up there in Milford, Kansas, did you plead guilty to a liquor case? *A.*—I did.

Q.—Did you pay a fine? *A.*—I did.

Mr. Morriss Sr.:—The witness answered before we could make the objection, and we didn't anticipate it. We make the same objection as to that.

THE COURT:—Isn't that charge made in the article?

Mr. Morriss Sr.:—It is not a charge in issue.

THE COURT:—The entire article is in evidence in the first place, and in the second place the question as to this man's reputation and the damages sustained, if any, by this article that is issued here—all of these matters that tend to show what his history and record are go to make up his reputation, and for that reason it is admissible in evidence, and for that reason I will overrule the objection.

Mr. Morriss Sr.:—We except to the ruling.

Q.—Were you arrested up in Kansas for shooting a man or shooting at him, or something of that kind—had a shooting scrape?

Mr. Morriss Sr.:—Same objection.

THE COURT:—I understand.

A.—No, sir. I didn't get arrested for shooting at a man.

Q.—You were arrested in some kind of a find or shooting scrape?

Mr. Morriss Sr.:—We make the same objection. There is no indication on earth who was right or wrong.

THE COURT:—Overruled.

A.—This fellow had me put under a peace bond because I made some remarks concerning him that caused him to be afraid, I guess, and they put me under a peace bond; I don't know whether I was arrested or not, but I had to give a bond to not shoot him.

Q.—As to the different specialties that you followed back there in Greenville, North Carolina, you were there, as I understand it, working as a doctor in venereal diseases, gonorrhea and syphilis, is that right? *A.*—I wasn't working at all, we were just getting the office opened up there, and that was the kind of office it was going to be.

Q.—And, then, you have advertised that you can treat cancer? *A.*—No, sir, I haven't advertised that I treat cancer.

Q.—That you had a cancer cure? *A.*—No, sir. What you are referring to is some experimental work that I was carrying out.

Q.—A cure on cancer—a cure for cancer? *A.*—Following the line of reasoning of Maud Slye of Chicago.

Q.—Have you advertised that your treatment that you have given in your practice will benefit cancer? *A.*—No, sir.

Q.—You haven't? *A.*—No, sir.

Q.—Do you advertise that your treatment benefits insanity, Doctor? *A.*—I haven't advertised in that sense, but I have reported cases of insanity that I have treated that have later recovered.

Q.—And, then, of course, you have many of these advertisements that your treatments aid in sexual rejuvenation, haven't you, Doctor? *A.*—Yes, sir.

Q.—We went over that this morning? *A.*—Yes, sir.

Q.—Now, your main specialty is the treatment of the prostate gland, is that right? *A.*—Yes, sir, the enlarged and infected prostate.

Q.—Doctor, this prescription 1020 that you treat your patients with, when you sell that prescription how much do you get for it? *A.*—I don't sell it in the way you mean it.

Q.—Well, I mean how much does it cost the person that is going to get it that comes to your hospital or that you use it on? *A.*—Well, it is not given to an individual separately, but it is always in combination bits, never given separately by itself.

Q.—If they get it, the patient, it costs them a hundred dollars, doesn't it? *A.*—That would depend altogether; some patients pay us a hundred dollars, but they get different kinds of medicine when they are paying that, and they may get the prescription 1020.

Q.—I am talking about 1020. *A.*—Yes, sir.

Q.—The 1020 prescription. *A.*—There isn't anybody that comes and pays us a hundred dollars and gets 1020.

Q.—Six tubes of 1020, don't you sometimes sell that for a hundred dollars? *A.*—No, sir; that wasn't the way it was handled; we used to treat the infected prostates whenever a man came here to the hospital.

Q.—I am just asking you now about selling it, and how much did you get for prescription 1020?

Mr. Morriss Jr.:—Counsel interrupts the witness, he asks him a question and then he interrupts the witness before he has time to complete the answer, if your honor please.

THE COURT:—Let him answer the question. However, I will admonish the witness to listen carefully and answer the question, and do not answer something else—and you will both save time.

Q.—Did you ever get any money when you gave a patient 1020, Doctor? *A.*—Most assuredly.

Q.—You did? *A.*—Yes, sir.

Q.—How much did you get? *A.*—Well, I can't answer you that question.

Q.—Now, what is in this 1020? *A.*—1020 is a number that goes on different ampules that I have in my hospital, and the ampules are numbered 1020, and those ampules always contain a certain amount of hydrochloric acid, one in a thousand, and the twenties indicate 20 cc. of liquid in the ampule, and the hydrochloric acid strength of the ampules is one in a thousand, and that is the reason for the 1020 being on that ampule; some ampules 1020 contain nothing but methylene blue water and hydrochloric acid, and other ampules contain hydrochloric acid and coloring matter and some of the iodides, and sometimes a small amount of mercury.

Q.—You mean that this 1020 has different things in it sometimes? *A.*—Yes, sir, there are varied forms; the idea of the 1020 is to signify a definite thing.

Q.—I thought 1020 would be the same thing, but I am so ignorant of such a matter. One 1020 will have one thing and another 1020 will have another thing, is that it, Doctor? *A.*—Yes, sir; and it is identified by the different colors; and there is 1020 blue and 1020 red and 1020 pink, and things like that.

Q.—What do you put in 1020 blue? *A.*—1020 blue is a solution of one in a thousand hydrochloric acid, methylene blue and water.

Q.—How much does that material cost you wholesale? *A.*—Oh, those ampules in large quantities, five thousand or ten thousand at a time, cost less than a quarter apiece, I think about eighteen cents.

Q.—You mean filled? *A.*—Yes, sir.

Q.—About eighteen cents apiece? *A.*—I think so; that is, if you buy them in large quantities, that is true.

Q.—Are those ampules that have the 1020 on them, are they blue (showing witness instrument)? *A.*—I don't know whether it was blue or what it was; I would have to see the color before I could tell you.

Q.—Those were your ampules? *A.*—I don't know whether they were or not.

Q.—Look at them there. *A.*—I am looking at them. I don't know whether they are my ampules or not.

Q.—Those labels are yours? *A.*—I don't know; I have no way of identifying them, the way they are there.

Q.—You can't tell whether they are yours or not, is that what you mean? *A.*—No, sir.

Q.—Where did you have those labels printed? *A.*—The labels that go on the ampules—we have the ampules manufactured in the laboratory at New York, and the manufacturing chemists label them in the laboratory.

Q.—Is there anything different in the labels that you see on there and used to be on your 1020 ampules? *A.*—You know, it has been so long since I looked at one of those labels, I don't know.

Q.—This hasn't got a cork in it, but it comes up here with glass and is twisted around and is air tight. *A.*—Yes, sir; and if you want to get it out you have to break the top.

Q.—You have to break the top? *A.*—Yes, sir.

Q.—Now, what do you do with the 1020 blue when you give it to a patient? And how does he take it inside of the body, how do you get it into his body? *A.*—It is given into a vein.

Q.—You inject it into a vein? *A.*—Yes, sir.

Q.—You don't inject it into a muscle? *A.*—No, sir.

Q.—After you inject it into the vein what effect does it have on the patient? *A.*—It increases the number of white blood cells.

Q.—Now, is this 1020 blue that you speak of, is that isotonic? *A.*—I don't know whether it is or not.

Q.—Do you know what isotonic means? *A.*—It escapes my mind for a moment; I don't grasp the meaning of it.

Q.—You don't know what isotonic means? *A.*—It escapes me for the moment.

Q.—Do you know what hypertonic means? *A.*—I can't frame a definition in my mind, no.

Q.—Would you read this, please (handing the witness instrument); this is a piece that appeared in the Bureau of Investigation pamphlet of the American Medical Association, a laboratory report, and see if that is a correct analysis of your 1020? *A.*—Well, I don't see anything in here saying anything about the presence of hydrochloric acid.

Q.—Well, you can turn it over there, it turns over, I believe. Yes, sir, there it is (indicating). *A.*—My solution contains methylene blue and hydrochloric acid, and I don't see that this says anything about the hydrochloric acid being in there.

Q.—All right, now, you heard Dr. Peterson yesterday describe in detail the way you performed the operation at the present time—it is Dr. Petermeyer, I beg your pardon. *A.*—Yes, sir.

Q.—You listened to that description? *A.*—Yes, sir.

Q.—Was it correct, his description of the operation correct? *A.*—In a general way, yes, sir.

Q.—How would you add to it, or how do you want to change it? *A.*—I would let it stand as it is. If I was describing the operation, why, naturally, my description would be a little different.

Q.—I don't want to take the time— *A.*—I will accept his explanation as satisfactory, in order to stay out of all of that rehashing.

Q.—Now, tell us, please, does a little goat gland have little nerves there when you take a little testicle out of a young goat, does it have nerves before it is taken out? *A.*—Naturally, any living organism in an animal body has nerves and blood vessels.

Q.—Now, explain to us, please, how you could make that little testicle of a little—how old were the goats ordinarily? *A.*—About three weeks.

Q.—That little testicle of a young goat, you claim that that lived and grew after you implanted it in a human testicle? *A.*—Some of them seemed to grow and enlarge and others, the majority of them, went through a process of absorption.

Q.—Absorption? *A.*—Yes, sir, they were gradually absorbed.

Q.—How long did it take to absorb? *A.*—I examined patients following the transplantation of them and some of them are gone in three months and some in six months, and I have seen evidence of remnants of them after even one and two years.

Q.—You mean to say that little thing lived and was just like a part of the human testicle, and it was living in there after you put it in there? *A.*—No, sir, I don't conceive of it that way, I don't conceive of it as being a part of the human testicle.

Q.—Why wouldn't it just get rotten in there? *A.*—If it became infected it would, it would spoil.

Q.—Well, if you put that little piece of meat in there and it is not getting any blood or anything, just laying in there— *A.*—You see, the patient's blood naturally bleeds into the little pocket where the gland is placed.

Q.—Where is it coming from if there isn't a hole in the blood vessel of the human for it to get out? *A.*—The gland itself is a sponge-like tissue, just like a piece of sponge, and whenever you drop it in a patient where there is an open wound it immediately becomes filled with the blood from the patient, just like a sponge becomes filled with water from a drop of water.

Q.—You don't claim you connected up the nerves or any blood vessels? *A.*—Oh, Lord, no.

Q.—You just took this little thing separate and put it in that slit in the testicle and sewed it up? *A.*—Yes, sir.

Q.—And the blood in the body of that testicle would squeeze in there to keep that thing alive so it wouldn't rot in there, is that the idea—I may express it very crudely. *A.*—Naturally when that foreign matter was placed inside the patient's body it was surrounded by the patient's blood vessels, and that would be the first thing that would happen and the blood vessel—I mean the patient's white corpuscles would naturally rush in and in the process of inflammation that would be set up in there in some of these patients there would be a substance like a sac that would really form around this gland; I have opened them up later and seen them where it looked like nature herself had tried to put a capsule around this gland.

Q.—Now, Doctor, just a question aside here. Now, up there in your country hospital, up there in Arkansas, the one out in the country, who now receives the patients there at the hospital, the doctor that admits them into the hospital? *A.*—Dr. Purcell.

Q.—How do you spell that? *A.*—P-u-r-c-e-l-l.

Q.—Now, in your advertisements or talks do you tell the patients or the public that you treat prostate glands without surgery? *A.*—No, sir.

Q.—You tell them you treat it without removing the prostate? *A.*—Yes, sir, that is it, yes, sir.

Q.—This part of your operation is that you take a tube and an electric needle and put it up there, and that is the surgery that cuts a part of the prostate off, isn't it? *A.*—Yes, sir, trims off part of the prostate.

Q.—Just briefly, without describing the operation, what do you claim is the effect—take a man that is suffering with prostate trouble and you operate on him with the usual and normal operation, what do you claim is the effect of that operation? *A.*—The prostate gland is reduced in size.

Q.—What is the effect that it has on the man? *A.*—The man feels better and he becomes more energetic and he gains weight and his appetite becomes better and his outlook on life is better.

Q.—Does it have any effect on his sexual powers? *A.*—Many of the patients say it has a good effect and the sexual powers are increased, yes, sir.

Q.—Sometimes does the patient, after he leaves you, for a post-operative treatment, and he goes home, does he take along with him some of the tubes of 1020? *A.*—No, sir; we don't permit that, and we haven't in several years.

Q.—You used to do that? *A.*—Several years ago we did, but we do not do that any more.

Q.—Well, now, when the patient took this 1020, when you used to let him take it home and let his own doctor treat him with it, these six tubes, how much did you charge him for it? *A.*—We wouldn't charge him anything for that; sometimes the man had to go home before he could complete the amount that we wanted to give him, so we would send the supply home with him so his physician could complete the treatment.

Q.—Didn't Dr. Petermeyer state that yesterday? *A.*—Dr. Petermeyer was confused there; we don't charge any patients a hundred dollars, and we don't send any medicine to be given hypodermically or intravenously to any patient.

Q.—I notice on one piece of literature it say "J. R. Brinkley, M.D.," and what is that M.D.? *A.*—Doctor of Medicine.

Q.—It says C.M., and what is that? *A.*—Master of Surgery.

Q.—Then it says Dr.P.H. *A.*—That is Doctor of Public Health.

Q.—Sc.D. *A.*—Doctor of Science.

Q.—And then "Chief Surgeon." Now, where did you get all of those degrees from? *A.*—Well, on this diploma I have, it says "Doctor of Medicine and Master of Surgery," and that entitles you to say M.D. and C.M. if you care to; the Doctor of Science degree was conferred on me by the Chicago Law School, an honorary degree, and the Doctor of Public Health was conferred on me by the Eclectic Medical University of Kansas City, Missouri, where I received my M.D. degree.

Q.—Who was that that conferred the Doctor of Science? *A.*—The Chicago Law School, Chicago School of Law.

Q.—So, it was the Chicago Law School? *A.*—Yes, sir.

Q.—Do you also put on your names sometimes LL.D.? *A.*—I will declare, I don't know.

Q.—Do you ever claim you have had a degree of LL.D.? *A.*—I don't remember.

Mr. Brown:—That is all the questions we care to ask.

Mr. Morriss Sr.:—We expect to put him on later, but we will not ask any questions now, Your Honor.

THE COURT:—All right.

Witness excused.

Mr. Reynolds:—Your Honor, at this time we would like to introduce a few exhibits, marked Exhibits 39, 40, 41 and 42, inclusive. (Handing instruments to counsel for plaintiff.) While those exhibits are being inspected, we are going to offer these in evidence, it being the marriage license of John R. Brinkley—well, if you wish to inspect those first, (handing instruments to counsel for plaintiff).

Mr. Brown:—Excuse me, Your Honor, but there was a matter that I overlooked to ask Dr. Brinkley.

THE COURT:—Do you want him to resume the stand?

Mr. Brown:—Yes, sir.

THE COURT:—All right.

Dr. John R. Brinkley, recalled as a witness for further cross examination, having been previously duly sworn, testified as follows, to wit:

FURTHER CROSS EXAMINATION

Questions by Mr. Brown:

Q.—Doctor, did you write these letters? (Handing witness instruments.) *A.*—Yes, sir, those letters were written by me.

Mr. Brown:—I offer the letters in evidence (Exhibits 43, 44 and 45).

THE COURT:—What is your objection to this, Counsel?

Mr. Morriss Sr.:—To these and the others, Your Honor, there are quite a number of different documents and there are various different objections to them, and we don't want to encroach upon the rule of discussion of the matter in the presence of the jury, and it brings up the question of some matters about which it will be necessary to put on some evidence.

THE COURT:—Particularly, what are the grounds, without the argument? There is a difference between making the argument and just giving the technical objection.

Mr. Morriss Sr.:—I shall show that the letter of February 8, 1935, is a privileged communication, and that, among others, is the objection. Then, as to all of them that they are immaterial and irrelevant to any issue, and they relate to a wholly different collateral matter not involved in any way in this proceeding, and they are incompetent to establish any issue or any defense in this case.

THE COURT:—All right, I will hear from the other side. I will ask Counsel, without testifying in any way or without attempting to state the contents of these things, to tell me why you think they are admissible.

Mr. Brown:—Your Honor, there is one part that is alleged on here by the plaintiff as to the effect that the doctor continues to make money, not withstanding the efforts of the various governmental departments and agencies, and there is another portion of the article declared on which says he is still on the air in spite of many efforts to remove him from the air, and these letters are written by Dr. Brinkley to the radio station.

Q.—Dr. Brinkley, was Mr. Burke your attorney? *A.*—Yes, sir.

Q.—And did you have a contract and have some business with Radio Station KFEG in St. Joseph, Missouri? *A.*—Yes, sir, I was broadcasting over it by remote control.

At this point there was considerable discussion over the introduction of pamphlets, documents and evidence relative to various incidents which were associated with the career of Dr. Brinkley and as to the possibility of the introduction of such materials.

EVIDENCE OF JACK K. TINGLE

Jack K. Tingle, official court reporter from Dallas, Texas, introduced in evidence copies of broadcasts made by Dr. Brinkley from his station in Mexico in 1931.

TESTIMONY OF DR. EUGENE W. SCHOEFFEL

Dr. Eugene W. Schoeffel, chemist employed by the American Medical Association, testified as to his examination of the contents of ampules sent to the American Medical Association by physicians who had obtained them from patients of John R. Brinkley.

The evidence proceeded:

Q.—Now, Doctor, will you tell us what you determined from your analysis to be the contents of these ampules.

Mr. Morriss Jr.:—The Court notes that our objection goes to all of this.

THE COURT:—All right.

A.—First the material was measured out.

Q.—Just a minute, Doctor, don't tell me how it was all done, but tell the jury what your conclusions were, what your findings were after all of these tests were made, the stuff that was in it, that is what we want to know. *A.*—You want to have the conclusion of the tests?

Q.—That is right. *A.*—The conclusion was placed down that this ampule contained approximately 20 cc. of a bluish- sapphire-like liquid. which had a density—I will have to quote,—

THE COURT:—You may refresh your memory, and I told you that, but you must not read it to the jury, but you can refresh your memory and then testify.

A.—Which had a specific gravity of 0.9985 in respect to water, and at 25 degree solution, 0.9971.

Q.—What do you mean by that last statement, just in ordinary language so everybody can understand it. *A.*—By specific gravity is meant the weight of a measured solution at a certain temperature.

Q.—And does your statement mean that it is about the same as water as to what you found as regards this specific gravity? *A.*—Something like it, very close to distilled water.

Q.—What else was found? *A.*—It was also found that the color of the solution is of the indigo type.

Q.—Will you explain just briefly why you use that term "indigo type"? *A.*—The color chemist knows approximately over 2,000 different blue colors.

Q.—And the indigo type? *A.*—The indigo type is a certain chemical formula by which various shades and hues of blue color can be made.

Q.—Is that the same coloring substance as one gets from methylene blue? *A.*—No, sir, it wouldn't be methylene blue, this is a different color type.

Q.—All right, now, what else was found? *A.*—Besides that, the spectroscopic analysis indicated the presence of several metal elements; however, these elements were found in traces only.

Q.—What do you mean by saying they were found in traces only? *A.*—From one of the tests we found that the solution contained approximately 0.004 of a per cent of dry material.

Q.—0.004? *A.*—0.004.

Q.—Is that in connection with these 20 cc. of material you say were in here after you had obtained that dry material in whatever process you used—could you see it with your own eyes? *A.*—It is an exceedingly small amount.

Q.—And what else was found? *A.*—Besides which we made tests for halogen, and that is the term for chlorine, iodine, fluorene and bromine.

Q.—What did you find? *A.*—0.2 cc. of the solution was tested with 1 cc. of diluted nitric acid and 1 cc. of silver nitrate test solution, and no detectable traces of chlorine could be found.

Q.—With that finding is it possible there was any hydrochloric acid in this solution, or not? *A.*—There is a remote possibility that chlorine could have been present if the entire 20 cc. would have been used.

Q.—And if chlorine were present that would mean hydrochloric acid, would it? *A.*—Not directly.

Q.—It might mean that? *A.*—It might mean.

Q.—From the sum of all of your tests were you able to determine whether or not, and have you a finding as to whether or not there was any hydrochloric acid in this solution? *A.*—I only can say that the tests produced on 0.2 cc. didn't give us a detectable trace of chlorine; however, it is known that a specialty ampule glass under certain conditions gives off traces of chlorine.

Q.—All right, now, Doctor, in making your analysis you spoke about finding dry substances, dry matters, dry materials, and how did you get that from this as it originally appeared? *A.*—The material was dried down in a platinum dish over a waterbath.

Q.—That is the same as boiling? *A.*—Boiling down.

Q.—And how large a speck did you find of dry material, the size of the head of a pin or the size of a pin point, or what—something we can understand. *A.*—Say, the point of a pin.

Q.—And it was from that dry material that all of your subsequent analysis was made. Is that right? *A.*—Yes, sir, after the acidity of the solution was stated.

Q.—What else did you find in the dry materials, if anything? *A.*—I have to refer back (looking at paper); the amount of hydrogen concentration we found to be of a very weak solution, and this led us to believe—

Mr. Morriss Sr.:—We object to what they would believe, Your Honor.

Mr. Reynolds:—I think he is going to—

THE COURT:—He can say what he believed himself, but he couldn't speak for anybody else. He used the term "us," and I don't know what he is talking about.

Q.—You made this analysis yourself, and that is what you are talking about? *A.*—Yes, sir, I do.

Q.—Well, what was it that you actually found, now, without regard to what you believed, at the time when you completed another test, just say what was it you found—I want to know what was in that pinpoint of matter.

THE COURT:—It seems to me the way you develop this is highly technical.

Mr. Reynolds:—It is very difficult.

THE COURT:—I don't know whether it is or not. State the technical proposition in order to get it into the record and then ask in terms of a, b and c what he is talking about. You asked Dr. Brinkley, or co-counsel did, and he said he had methylene blue and a little hydrochloric acid in it and outside of that it was water; now, you can take one of these chemists, one of these ten chemists, and he could probably talk to us about that for a week, take a little pinpoint of solid matter and break it down and talk about it a long time and nobody would understand it, and you are not getting anywhere.

Q.—The last question is to tell us, just enumerate the substances you found in that little point of a pin of matter, if you haven't named them all. *A.*—Traces of metallic elements, approximately 0.003 of a per cent of indigo type dye, and most of the indigo type dyes contain a small, practically immeasurable amount of sodium chloride, and the ampules also give off sodium chloride.

Q.—Did you find this substance to be an isotonic substance? *A.*—No, sir, the substance wasn't isotonic.

Q.—Will you explain to the jury, so that we can understand it, what we mean by isotonic? *A.*—Isotonic is understood to be a solution which is comparable with a 0.9 per cent sodium chloride solution, and it is generally called a physiological salt solution, and if the solution is below the content of 0.9 sodium chloride, the solution is called hypotonic, and if it is above 0.9 per cent sodium chloride it is called hypertonic.

THE COURT:—Now, wait a minute. The province of the Court in presiding over the trial is to proceed in order to get something out of it, and you asked him to explain that so we could understand it, and, after all, I suppose the purpose of putting the witness on was to find what he found in the ampules and say it so somebody besides a chemist can understand it, and if the ampule had medicine or chemical properties that had some significance, let him say so, and if it was nothing but colored water, let him say so, but a man can talk about H_2O and he may know what it is, but the general public wouldn't know it is water; so, let's talk in the terms of the Court and the Jury.

Mr. Reynolds:—He can't go into the therapeutic effects because it takes a doctor to do that, and we will tie that up later.

The cross examination brought out the fact that the ampules had been handed to the witness by Dr. P. N. Leech, director of the laboratory of the American Medical Association.

(To be continued)

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

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SATURDAY, MAY 13, 1939

THE WAGNER NATIONAL HEALTH BILL

In the Organization Section of this issue of THE JOURNAL appear statements of hearings held before the Senate Committee on Education and Labor last week, before which appeared Senator Wagner himself, Miss Josephine Roche and later Dr. Arthur W. Booth, representing the Board of Trustees of the American Medical Association. Previously representatives of many farm agencies had appeared before the Senate committee urging legislation to increase federal expenditure for health. At a subsequent date representatives of some of the various state medical societies will appear before the Senate committee, as will also a group of representatives of the American Medical Association.

Easily apparent thus far is the failure of the Interdepartmental Committee to Coordinate Health and Welfare Activities to be influenced to any considerable extent by the various agencies which appointed committees to consult with the committee. When the President of the United States sent the National Health Program to the Congress in January, recommending careful study rather than enactment, it was apparent that the National Health Program had not been modified in any appreciable manner as a result of such hearings. Moreover, in her statement before the Senate committee last week Miss Roche again emphasized the assertion that the statistics in the National Health Survey had not been challenged, notwithstanding the fact that in THE JOURNAL, as well as in many other publications, such challenges have been made. Especially notable, of course, are the considerations of the American Hospital Association and the Catholic Hospital Association, which certainly have challenged some of the statements relative to the needs in the hospital field. Indeed, the most significant challenge yet made appears in the words of Alphonse M. Schwitalla, S.J., who says "If all this is social security, if all this is necessary for the preservation of the national health, then justly a philosophy of health care must be restated, and medical practice and hospital practice as today understood must be razed to give way to overpowering structures of huge bureaus, national councils and state organizations founded not upon Christian inspiration but upon legal enactment." If any further challenge is needed, readers of THE JOURNAL are referred especially to the general statement presented by Dr. Arthur W. Booth, Chairman of the Board of Trustees, which appears on page 1972 of this issue.

The representatives of the American Medical Association who will appear before the Senate committee at a later date will present facts and documents which controvert absolutely some of the statistics and data on which many of the recommendations in the Wagner National Health Bill are based. The medical profession will do well to bear in mind that the House of Delegates of the American Medical Association at the session to take place in St. Louis next week no doubt will express clearly the point of view of the American Medical Association in this proposed legislation.

THE CALL FOR THE PHARMACOPEIAL CONVENTION OF 1940

The President of the U. S. Pharmacopeial Convention is authorized to issue a call for the decennial meeting on May 1 of the year preceding the meeting. By this time various medical societies and medical colleges, as well as various pharmaceutic groups, should have received the call and the request to send delegates. Before the Pharmacopeial Convention of 1930 the Council on Pharmacy and Chemistry[1] urged all organizations, medical, pharmaceutic, chemical and others, carefully to select delegates noted for high ideals, for breadth of vision and for sound judgment as well as for technical knowledge. The aims and purposes of the Pharmacopeia were admirably expressed by its principal founder, Lyman Spaulding, M.D., in the preface to the first edition.

In its editorial of ten years ago THE JOURNAL stated:

> Although federal laws concerned with the control of the purity of drugs provide for the enforcement of drug standards set forth by the Pharmacopeia, the book is not published by or under the control of the federal government. It is published by authority of the United States Pharmacopeial Convention. This body is composed of members who are sent as delegates by national or state associations of physicians, pharmacists and other groups concerned with medicine and drugs, by schools of medicine and of pharmacy, and by certain government services. The convention meets once every ten years, and its chief function is the selection of the Committee of Revision of the United States Pharmacopeia. To this committee is assigned the task of making any desired changes in the Pharmacopeia then in force and of issuing a revised edition of the book. The next Pharmacopeial Convention has been called for May 13, 1930, at which time the delegates appointed by the constituent bodies will meet in Washington, D. C., and inaugurate the preparation of the next, the eleventh, revision of the United States Pharmacopeia.

The convention of 1930 was in many respects unsatisfactory because its conduct was undemocratic and

1. The Call for the Appointment of Delegates to the United States Pharmacopeial Convention, J. A. M. A. **93**: 989 (Sept. 28) 1929.

unscientific. Astute and industrious interests overwhelmed the convention with their representatives. Fortunately, the Board of Trustees of the U. S. Pharmacopeia, the chairman and his helpers on the Revision Committee were able to carry out their work efficiently. As a result, the Pharmacopeia of 1930, which became official in 1936, was much better than might have been anticipated but it was produced without some assistance which might otherwise have been secured.

The entire method of developing the Pharmacopeial Convention requires radical revision. At present any organization entitled to representation may send three delegates. Medical organizations, unfortunately, do not feel in a position to pay the expenses of three delegates. Some schools of pharmacy and pharmaceutic organizations have full representation because commercial interests are willing to see that delegates participate in the convention. Thus in 1930 medical delegates were outvoted by this arrangement.

When the Pharmacopeia was first devised, distances were great and communications were difficult. It was therefore provided that no one could be a member of the Revision Committee unless he attended the Pharmacopeial Convention. The Revision Committee of 1930 was deprived of the services of many good men, had they been willing to serve, on no other ground than that they had not attended this haphazard, ill conducted convention. Medicine has asked either that it be given equal representation on the Revision Committee or that, as it requested in 1930, it be given one third representation, with the understanding that therapeutic scope shall be controlled entirely by the medical group and that questions of pharmaceutic necessity be decided by the pharmaceutic group. This was denied. If the cooperation of the medical profession is to continue, some effort must be made to meet the reasonable demands set forth in 1930.

In various meetings of the Council on Pharmacy and Chemistry, the Board of Trustees, and other bodies of physicians, the following principles have been formulated: To correct the evils of one-sided representation it would seem that only one vote should be accorded an organization. This would preclude the practice by some pharmaceutic interests of securing credentials from various organizations or schools and selecting delegates who will vote as they request in the convention. The rules should be corrected so that the most capable men in the country may serve on the Revision Committee, and the prerequisite that a man must attend the convention to serve on the committee should be removed. Until these amendments are made there should be restored the agreement of the 1920 Pharmacopeial Convention that medical men shall control therapeutic scope and pharmaceutic men shall control pharmaceutic scope.

During the past five years progressive leaders in the pharmaceutic field have shown commendable effort in trying to work out some method whereby a reasonable scheme of cooperation with physicians may be established. The Board of Trustees of the U. S. Pharmacopeia met recently with the Board of Trustees of the American Medical Association to discuss means of cooperation. Many physicians believe that the time has come when the Pharmacopeial Convention must decide on the general principles that have been mentioned before they will commit themselves to full participation on the various committees of revision. The medical profession has shown its desire to be cooperative. THE JOURNAL has published during the last year a series of articles on "The Pharmacopeia and the Physician." These articles were published at the request of the Pharmacopeia authorities and they have been received with much favor by both the medical and pharmaceutic professions. To the Pharmacopeial Convention of 1940 only those delegates, medical or pharmaceutic, should be sent who will come with broad-minded attitudes and with the serious wish to work harmoniously toward constructive policies.

Current Comment

SEX HORMONES HOLD THE STAGE

Today the desks of physicians are being flooded again with fantastic advertising claims for sex hormones. New heights have been reached with the publication of two pamphlets, "Perandren" (Ciba) and "Roche Review" (Roche-Organon). The recommendations reveal a policy with little regard for scientific evidence. The name of one of the Roche-Organon preparations, Neo-Hombreol (presumably new manhood oil), is significantly reminiscent of "Lost Manhood" exploitation. And most of the claims are apparently based on one or two experiences in which the reporter has neglected to discount for the power of suggestion. Ciba naively advises testosterone propionate for therapy on the basis of admitted "bits of evidence, published and unpublished." "Atonicity" and "mammary gland pathologies" are among the firm's recommended indications for androgenic therapy. But this is conservatism itself when compared to the opus of the "scientific staff" of Roche-Organon, who have prepared a chart and "dosage schedule" for sex hormones. Here are indications, dosage and everything except references to scientific literature urging androgenic therapy for many conditions, including epispadias, hypospadias (conditions which always have required and always will require surgery) and Graves' disease in women. Estrogenic therapy is prescribed for acne, alopecia, impetigo, Buerger's disease, iridocyclitis, polyarthritis and many another condition. And in the following effusion Mr. Elmer Bobst really "goes to town."

Dear Doctor:

No DOUBT young Juan was greatly admired by all the young ladies of Servas as he matured, for he was a fine looking lad, with a splendid physique and possessed a daring personality. His adventuresome spirit forced him at a very early age to

take a hand in the Moorish Wars, and he was only 33 years old when it led him again to persuade Columbus to let him go along on his second trip to the new world. But by the time the estimable Juan had conquered the Island of Porto Rico and had settled down to the rather soft and engaging life of its Governor, the first shadow of old age started to fall across his pathway. As his vigor began to wane, the Indian legends about the Land of Bimini and its wonderful spring of waters with their marvelous curative powers intrigued him more and more. And soon thereafter Juan Ponce de Leon—for it was none other than the famous Ponce de Leon—set forth on his memorable search for the so-called Fountain of Youth. When Ponce de Leon began his quest of the youth-restoring waters, he was just about fifty years of age. Yes, just about the period of man's life when the downhill journey becomes significant. Is it due to the rapid decrease of the male sex hormone secretion at middle life? More and more physiologists and endocrinologists are coming to that conclusion. A study of the clinical characteristics of the male climacteric, embracing physical, psychological, and functional changes, leads rather definitely to the conclusion that proper administration of testosterone propionate can play the same replacement role in man as does estrogen therapy in the menopause, or female climacteric. Testosterone propionate, which we are making available under the name of Neo-Hombreol, is the active, chemically pure, crystalline propionate salt of the male sex hormone, identical in every respect with the same substance derived from natural sources and with the added advantage of synthetic production. We suggest the experimental use of Neo-Hombreol for combating the annoying symptoms of the male climacteric. Treatment should be started with injections of Neo-Hombreol. For maintenance purpose we then suggest the use of Neo-Hombreol Ointment which is now available in a most accurate dosage form called the Dosule. We will gladly send you a 3-day supply of Neo-Hombreol Dosules if you will make use of the enclosed card.

Sincerely yours,

Elmer H. Bobst,
President.

This flamboyant exploitation obviously presupposes an extraordinary degree of ignorance on the part of the physician. It can do little more than delay the scientific use of these compounds, meanwhile classifying the concerns responsible with others who sponsor reckless claims. The report of the Council on Pharmacy and Chemistry on the Present Status of Testosterone Propionate and on rejection of Perandren, Oreton and Neo-Hombreol appears in this issue of THE JOURNAL, page 1949.

SULFANILAMIDE AND SMALLPOX

Elsewhere in this issue (page 1936) is a report of the treatment of four cases of smallpox with sulfanilamide. In three there was only an evanescent macular eruption which rapidly disappeared, and in the remaining case there were only three pustules. Three additional patients observed about the same time were treated only symptomatically and in all three the typical eruption of smallpox developed. All treated and untreated patients recovered. This interesting observation seems to indicate that sulfanilamide, when given early in the disease, has a beneficial effect on the cutaneous lesions and possibly on the disease itself. Although smallpox is increasing (there were 14,355 cases reported for the year 1938) the disease is now mild, as evidenced by only thirty-five deaths in 1936 out of 7,459 cases in that year. In the presence of such a low case fatality rate, conclusions as to the value of sulfanilamide in preventing mortality from smallpox cannot be drawn from the four cases thus treated.

ABSORBABILITY OF CATGUT

Two recent studies on the absorption of catgut are of much practical importance to surgery. Bates [1] made experiments on adult dogs, using various sizes and types of catgut. After varying intervals it was found that plain catgut excites a prompt and violent exudative foreign body reaction which delays the appearance of fibroblasts and therefore delays wound healing. Large, plain catgut however was absorbed about as fast as the smaller sizes, but no size held throughout the time during which wound support is needed. Chromic catgut was associated with a retarded and lessened exudative foreign body reaction and with the early appearance of fibroblasts and early healing. Further, Bates found that small chromic catgut functions longer than large chromic catgut and that wound support and healing were most satisfactory when the smallest chromic suture was employed. In experiments on human beings, Wolff and Priestley [2] reported tests of the rate of absorption of various types of catgut. On closure of the incision in an unselected series of abdominal operations an interrupted suture of the catgut to be tested was placed through the anterior fascia of the rectus muscle and tied round a loop of nonabsorbable suture material which was permitted to project from the skin. Gentle traction was made on the projecting suture material every day or two after operation until it came out, thus indicating that the catgut was absorbed. In many cases three and four of these test sutures were used simultaneously. Three hundred and fifty-eight strands of catgut were tested on 164 patients. It was found that small sizes of catgut remained unabsorbed as long as or longer than larger sizes. The average absorption time of 000 chromic catgut in one series of patients was fifteen days. In contrast, strands of chromic catgut 1 and 2 of the same brand were absorbed in the average time of 10.6 days. Single strands of catgut lasted as long as double strands. It might be concluded, therefore, that the only advantage in the use of double strands is to secure greater initial tensile strength. In certain cases chromic catgut was absorbed as fast as or faster than plain catgut, although as a rule the process of chromicizing catgut materially lengthens the time necessary for its absorption. Great individual variations in absorption time were found, the greatest when chromicized catgut was used. These investigators further concluded that labels which indicate the length of time necessary for absorption of catgut are entirely fallacious as far as the human being is concerned, that drainage and suppurating wounds do not cause early absorption of catgut, and that certain brands of catgut consistently last longer than other brands. The practical applications of these newer studies of the absorbability of catgut deserve early confirmation and wide attention.

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1. Bates, Robert R.: Studies on the Absorbability of Catgut, Am. J. Surg. **43**: 702 (March) 1939.
2. Wolff, L. H., and Priestley, J. T.: The Absorption of Catgut in Human Beings, Proc. Staff Meet., Mayo Clin. **14**: 149 (March 8) 1939.

ORGANIZATION SECTION

HEARINGS ON SENATE BILL 1620, THE SO-CALLED WAGNER NATIONAL HEALTH BILL

On May 4, 1939, Senator Robert F. Wagner appeared before the Senate Committee on Education and Labor to present a statement on the National Health Bill. Briefly, Senator Wagner called attention to previous legislation for health, including the Sheppard-Towner Act, the Social Security Act and the legislation establishing the National Cancer Institute. He pointed out that the President had sent the National Health Program with its considerations to Congress for further study.

Senator Wagner pointed out that the costs of medical care have increased as our resources of medical skill, facilities and personnel have advanced, but that inequalities in the distribution of medical care have been widened. He also said that the National Health Bill was drafted in response to a plea from the people that our government take health from the list of luxuries to be bought only for money and added to the list containing the inalienable rights of every citizen.

Mr. Wagner stated that there is no intention to put the federal government into the business of furnishing medical care and that on all the phases of the bill the initiative will rest with the states. Further, that federal encouragement and cooperation will be effected through the traditional method of grants in aid. He then explained the various phases of the bill. In association with preventive measures is a new program, under title XIII, to enable the states to extend and improve existing provisions for the care of the sick and the treatment of disease. He said: "The Social Security Board, which is charged with the administration of this title, is not authorized to give or withhold grants at pleasure, or to require state plans to develop along any predetermined lines. I have said again and again that nothing in the bill requires the states to establish compulsory health insurance. Within the limits of certain necessary standards, the states are free to provide programs of medical care compatible with the needs and desires of their own people, and made effective through methods of their own choice. The state plan may apply merely to people on relief or extend to others higher in the economic scale. It may be financed by general taxation, by the contributions of the beneficiaries of the medical services, or by combinations of these methods. The scope of medical services and the manner of their provision will be for the individual state to decide, so long as the program is statewide or becomes such within five years, involves financial participation by the state, and complies with other standards prescribed by the bill."

Senator Wagner said that the bill does not propose to flood the country with hospitals and health centers, and that no money would be advanced until the submission of a careful state plan. He also defended the reorganization which places the United States Public Health Service under the Social Security administration with the following statement:

"The plan of reorganization recently submitted by the President properly conceives of health protection as an integral part of our efforts toward human security. By placing both the Social Security Board and the Public Health Service in the new Federal Security Agency, the reorganization plan establishes a sound nucleus for the coordination of federal health functions which is so essential to the efficient operation of a national health program."

It was his concept that the exactment of this program would result in the practice of economy, since sickness is responsible for one third of all dependence on public relief or private charity.

"In closing," he said, "I want to pay deserving tribute to the unselfish spirit, enterprise and generosity of the members of the medical profession for the splendid and self-sacrificing labors in alleviating sickness and promoting the people's health. A major purpose of this legislation is to help them to carry more effectively a burden which they have long carried alone. Both in the consideration of the bill and in its administration upon enactment, we bespeak, and we know we will have, the support and cooperation of the members of the noble profession which 'has for its prime object the service it can render to humanity.'"

STATEMENT BY JOSEPHINE ROCHE

Miss Josephine Roche, in her appearance before the Senate Committee on Education and Labor, recapitulated the development of the National Health Program and told the story of the National Health Conference. She emphasized also the deficiency of medical care among the poor and among people with small income, and some of the other statements made in the National Health Survey. In her statement on the National Health Conference she said "At the end of the first day's session, one of the participants asked the question 'Are there any members of this conference who seriously challenge the statements with reference to need which were made this morning?' This question was stated to the conference by the presiding officer. There were no challenges then and there have been none since." Obviously all readers of THE JOURNAL know that the figures of the National Health Survey as to medical care have been repeatedly challenged in many ways by many different agencies.

Miss Roche then proceeded to outline the National Health Program as developed by the Interdepartmental Committee to Coordinate Health and Welfare Activities of the United States government. She then pointed out that her committee did not recommend state medicine or a federally administered system of compulsory health insurance but that it did recommend the assistance of grants in aid. She stated that nineteen groups had met with her committee following the National Health Conference and that these groups were essentially in agreement as to the need for action and, "so far as there were any differences of opinion among some of these groups, they differed only as to how far and how fast such action should proceed." In her conclusion she said:

"May I emphasize that the committee's recommendations are not aimed merely at conditions as they exist today but, in the words of the President of the United States to the National Health Conference, 'we have before us a comprehensive, long-range program, providing for the most efficient cooperation of federal, state and local governments, voluntary agencies, professional groups, mediums of public information and individual citizens.' The committee does not visualize that it is practicable to put into effect immediately the maximum recommendations. It contemplates expansion along well planned lines with a view to achieving operation on a full scale within ten years. However great the difficulties, it must be realized that we shall learn only by doing. The committee therefore feels very strongly that the time has come to make a decision on a national health policy and a beginning on a national health program.

"The bill introduced by Senator Wagner, S. 1620, now under consideration by the Senate Committee on Education and Labor, would translate into reality the recommendations of the Interdepartmental Committee. In view of the importance of the nation's human resources, I trust that this legislation may be given serious and prompt consideration."

STATEMENT OF DR. ARTHUR W. BOOTH, CHAIRMAN OF THE BOARD OF TRUSTEES OF THE AMERICAN MEDICAL ASSOCIATION

On May 5, Dr. Arthur W. Booth, chairman of the Board of the Trustees of the American Medical Association, appeared in behalf of the Association and made the following statements:

I thank the committee for this opportunity to state the position of the American Medical Association with respect to some of the proposals set forth in the pending bill, S. 1620, and for the opportunity to be given later to introduce evidence in support of the Association's position. In order that the committee may better appraise what I say and the evidence to be presented hereafter by the Association, I submit for the record a memorandum showing briefly what the American Medical Association is and what its objectives are. Such information is particularly needed just now because of attacks on the Association, some made in good faith and some made apparently because of selfish interests.

It is sufficient at the present moment to say that the American Medical Association is a federation of state medical asso-

ciations. It is governed by a House of Delegates, made up of 154 delegates elected by the state medical associations of which the American Medical Association is composed; fifteen elected by the several sections into which the Scientific Assembly of the Association is divided, each devoted to a particular branch of medicine, such as surgery, obstetrics and internal medicine; and three appointed, one each by the Surgeon General of the Army, of the Navy and of the Public Health Service from the commissioned medical corps of those services. Of the 173,879 physicians lawfully authorized to practice medicine in the United States, 113,113 were on May 1 of this year members of the Association.

The House of Delegates of the American Medical Association fixes the policies of the organization. The House has not met, however, since the bill now under consideration was introduced. It will assemble on May 15 in St. Louis and will consider the bill, and representatives of the Association will report to your committee as promptly as practicable whatever action the House takes. They will lay before you, too, some of the many data that the Association has assembled during recent years, relative to the economics of medical service from the standpoint of the physician, of the hospital and of the people.

Although the House of Delegates has not had an opportunity to consider the pending bill, it did consider, at a special session in September last, the so-called National Health Program formulated by what is sometimes referred to as "The Technical Committee on Medical Care." This Technical Committee, it should be understood, was created by a committee appointed by the President and known as "The Interdepartmental Committee to Coordinate Health and Welfare Activities." The report of the Interdepartmental Committee to the President, including the report of its Technical Committee, was submitted to the Congress by the President [See H. R. Doc. 120, 76th Congress, 1st session], and S. 1620 proposes to make the recommendations of the Interdepartmental Committee, or some of them, effective. The conclusions of the House of Delegates of the American Medical Association, at its special session in September last, based on the report of the Technical Committee, are therefore relevant to the pending bill and to the deliberations of your committee, so far as the bill is founded on that report.

Judged by the principles laid down in the resolutions of the House of Delegates in September last, S. 1620, as a whole and in many of its details, is unsound, and its enactment would not be in the public interest. Its enactment, it is believed, would prove a costly mistake, jeopardizing the welfare of the people and seriously compromising the future development of the science and art of medicine in the United States. If the federal and state taxes that will have to be imposed to carry this bill into effect must be imposed, then at least a substantial part of the proceeds had better be devoted to providing for the needy adequate food, clothing and shelter. So far as this bill is concerned, hunger, nakedness, cold and storm are to be left to wreak their damage, the bill proposing only to provide medical, hospital and nursing service to cure the damage for a while after it has been done.

If the principles laid down by the House of Delegates are unsound, the Association will welcome enlightenment. Fortunately, the proponents of this legislation have the opportunity of presenting their evidence in support of it before the House of Delegates of the Association meets in St. Louis, May 15-19. That will make it possible for the House to have such evidence before it in the course of its deliberations. If the House that is about to convene finds that modifications of the conclusions of the House that met in September last are called for, it will make such modifications.

Coming now directly to a discussion of the pending bill, I call attention to the fact that it proposes to authorize the imposition on the people of annual federal appropriations of variable amounts, ranging from $98,250,000 upward to an indefinite skyward limit and to require the states that cooperate with the federal government under the act to impose on their people proportionate state appropriations. It may seem paradoxical to speak of "imposing" appropriations on the people, but since every appropriation has implicit in it the taxes that have been or must be levied to provide the money appropriated, every appropriation that is made is "imposed" on the people just as truly as are the taxes themselves. The fact that an appropriation is primarily for the benefit of one class of the population while the taxes necessary to cover the appropriation fall directly on another class, possibly even on future generations, does not alter the situation.

So far as I am informed, no evidence has ever been offered to show that any of the projects embodied in S. 1620 were devised by any of the people of the several states or that the enactment of this legislation is being promoted by them. On the contrary, it is understood that every project embodied in this bill was devised by appointive federal officials and employees, to be handed down to the people on a "take it or leave it" basis. If the bill is enacted, the federal government will levy on the people the taxes necessary to provide for the payment of the cost of all activities authorized under the act and will then say to the people "Unless you come in on this venture, we keep every cent we have taken from you." Propaganda has been and is being organized to advance the plans of the federal proponents of this legislation, but the federal origin and driving force back of all these projects must not be lost sight of. The origin of these projects need not damn them, but it certainly lays on their federal organizers and proponents an even heavier burden of justification than they would have if the projects had originated in the several states. In meeting that burden, inasmuch as the taxes that the bill requires will fall on the people of all the states, its proponents are called on to justify it for each and every state; not for Alabama or Mississippi or Nevada alone but for New York, Pennsylvania, Massachusetts, Illinois and every other state. The prime necessity for such a state by state justification of this bill lies in the fact that, if there are states whose people are destitute and in distress beyond the power of their respective states to aid them, the Congress may appropriate for their relief on such terms as it sees fit, but without using such destitution and suffering in one state as an excuse or justification for imposing tax burdens and federal guidance and control on states whose people are not destitute or suffering and that are able to manage their own affairs.

It has been claimed in support of universality for legislation of this type that the federal taxation and the federal guidance and control that go with it are necessary:

1. To stimulate the states,
2. To equalize the health opportunities in all the states, and
3. To procure uniformity in health activities throughout the United States.

To lay the heavy hand of federal guidance and control on a state may serve quite as well to destroy its initiative as to stimulate it, and to subject every state to federal control and thus to establish uniformity may deprive the federal government of stimuli from the states; and stimuli from the states and a critical attitude on their part are as necessary to federal efficiency as are federal stimuli and criticism needed to insure state efficiency.

The phrase "to equalize the health opportunities in all the states" is a seductive phrase, but what assurance have we that the increase in taxation and the establishment of federal supervision and control to bring health opportunities to a common level, while raising the opportunities in some states, will not degrade the opportunities that exist in others and thus procure equality, certainly, but an equality of doubtful advantage?

To bring about uniformity of health activities among all the states would certainly tend to delay and possibly to diminish and to destroy opportunities in health endeavors for variation and experimentation that is possible under state control alone, which, under our normal constitutional form of government, furnish advantages that are too little appreciated and taken advantage of. The House of Delegates of the American Medical Association, at its special session in September last, emphasized particularly the need for the promotion of local initiative, supported by state aid and guidance if necessary, and by federal aid, guidance and control only when rendered necessary by the destitution or incapacity of the state.

The fact that the authority granted certain federal officers and the money placed at their command, by the Social Security Act, have not already reduced illness and disability to the extent apparently anticipated by some of the proponents of that act seems to have led to the demand in this bill for enlarged authority and more money, as if authority and money were together all sufficient to work miracles in the field of health. If this bill is enacted, in all probability within a year or two we shall have a repetition of our present experience, a demand for more authority and more money, in the vain hope of hastening a millenium in the field of health. But there is no royal road to health, either of the people as a whole or of individuals. Time and effort are required over long periods of years, and, even after such long periods, time and effort will be found to have produced smaller returns in some communities than in others; for conditions over which we have but limited control, such as race, heredity and meteorological conditions, may be the chief determining factors in causing diseases and death.

If the federal government can, in the brief ten years that has elapsed since the Sheppard-Towner Maternity and Infancy

Act was expressly repealed by the Congress, June 30, 1929 [Public No. 566, 69th Congress; 44 Stat. L. p. 1024], go as far as is proposed in this bill in spreading itself over intrastate activities in the field of health and medical service, there would seem to be no limit on its right to expand. A federal subsidy for police service might well give the federal government supervision and control of all police service throughout the country. A federal subsidy for the operation of state systems of assessment and tax collection might be devised to give the federal government supervision over state activities now regarded strictly as state functions. Possibly a line might be drawn against federal encroachment on the legislative activities of a state, but one can readily envisage a federal subsidy for the aid of state legislatures that would lead to the federal direction and control of their functions.

Concerning the action of the House of Delegates of the American Medical Association in special session in September last, there has been some misunderstanding and some misrepresentation. To make clear what the House did, it is necessary to go at some length into its proceedings. In doing so it is necessary to emphasize again the fact that the action of the House of Delegates at that time had reference not to S. 1620 but to the recommendations of the Technical Committee on Medical Care, an agency of the Interdepartmental Committee to Coordinate Health and Welfare Activities created by the President on which that bill seems to rest. It is therefore only as S. 1620 undertakes to translate into organization and action the recommendations of the Technical Committee that the action of the House of Delegates in September 1938 is pertinent.

1. The House of Delegates in September 1938 recommended the establishment of a federal department of health with a secretary at its head, who should be a doctor of medicine and a member of the President's cabinet. S. 1620 provides for no such officer. In fact, it proposes to leave the preventive and curative medical services of the federal government scattered as widely as ever through the federal organization.

2. The House of Delegates proposed that expenditures for the expansion of public health and maternal and child health services should not include the treatment of disease except so far as it could not be successfully accomplished through the private practitioner. The pending legislation purposes to provide for the treatment of disease under various conditions, without in any way safeguarding the existence of the private practitioner.

3. The House of Delegates favored the expansion of general hospital facilities where need exists and emphasized its approval of the recommendation of the Technical Committee stressing the importance of the use of existing hospital facilities. The House of Delegates did not endorse the committee's recommendation for an increase in special hospitals and for what the Technical Committee calls "health and diagnostic centers." S. 1620 includes no provision for the increased use of existing hospital facilities. It does not limit the expansion of general hospital facilities to situations in which need exists. It does propose an extensive program in the field of special hospitals and health, diagnostic and treatment centers.

4. The House of Delegates advocated recognition of the principle that the complete medical care of the indigent is a responsibility of the community and of the medical and allied professions and that such care should be organized by local units and supported by tax funds. It recognized the necessity for state aid for medical care in poorer communities. It proposed the limitation of federal aid to cases in which the state was unable to provide assistance for local communities. S. 1620 proposes to make federal aid for medical care the rule rather than the exception. Nothing has been found in it to limit to persons in need the medical care provided by federal funds.

5. The House of Delegates urged the importance of well coordinated programs for the improvement of food, housing and other environmental conditions for the prevention of disease and the promotion of health. It urged the establishment of a definite and far reaching public health program for the education and information of all the people, to enable them to take advantage of the medical service now available. For these basic needs for the prevention of disease and the promotion of health, S. 1620 makes no provision.

6. The House of Delegates refused to foster any system of compulsory health insurance. S. 1620 proposes to pave the way for compulsory health insurance by leaving to the Social Security Board the determination of what constitutes "methods of administration" necessary for the efficient operation of state plans for medical service. The provisions of the bill for grants to states for temporary disability compensation squint in the same direction. The House of Delegates approved the provision of compensation for loss of wages during sickness, but S. 1620 proposes to provide not merely for loss of wages but to provide medical services also.

7. The provisions of S. 1620, authorizing the chief of the Children's Bureau and the Surgeon General of the Public Health Service to set up what are termed "demonstrations," the provisions for the training of personnel, the provisions for an indefinite number of ill defined "advisory councils" and the provisions for the promulgation by various agencies, federal and state, of rules and regulations are matters that have never come before the House of Delegates of the American Medical Association. Obviously, they are all of serious import and will require careful consideration.

The House of Delegates authorized the appointment of ten physicians, under the chairmanship of Dr. Irvin Abell, President of the American Medical Association, to confer and consult with the proper federal representatives relative to the proposed National Health Program. This committee was not consulted in the framing of the pending bill. Had it been consulted, it is possible that through joint studies and investigations and mutual endeavor a bill would have been produced containing an irreducible minimum of issues between the medical profession of the country on the one hand, so far as it is represented by the American Medical Association, and, on the other hand, the proponents of this legislation, a consummation for which your committee might well most devoutly wish.

MEDICAL LEGISLATION

MEDICAL BILLS IN CONGRESS

Changes in Status.—The President on April 25 submitted to Congress Plan No. 1 on government reorganization. He proposed, among other things, to set up a new agency to be known as the Federal Security Agency, in which will be grouped the Social Security Board, the United States Employment Service, the Office of Education, the Public Health Service, the National Youth Administration and the Civilian Conservation Corps. At the head of this new agency is to be appointed an administrator with a salary of $12,000 a year. Representative Taber of New York introduced a concurrent resolution in the House, H. Con. Res. 19, proposing that the Congress reject the President's plan. This resolution was defeated by the House of Representatives May 3, and the President's Plan will become effective on the expiration of sixty calendar days after the date of its submission. S. 685 has passed the Senate, proposing to establish in the United States Public Health Service a Division of Water Pollution Control. S. 1416 has passed the Senate, proposing to make the provisions of the United States Employees' Compensation Act applicable to civil officers of the United States. S. 1964 has been reported to the Senate, proposing to authorize national banking associations to make charitable contributions. H. R. 2987 has passed the House, proposing to compensate any employee of the United States government who shall furnish blood for transfusion to a member or former member of the military establishment who is a patient in a government hospital.

Bills Introduced.—H. R. 5763, introduced by Representative Barry, New York, proposes to authorize national banking associations to make charitable contributions. H. R. 6068, introduced by Representative Knutson, Minnesota, proposes that on all ethyl alcohol withdrawn and used for other than beverage purposes the tax shall be $1.10 per gallon. S. J. Res. 107, introduced by Senator Sheppard, Texas, proposes to authorize the President to award the Congressional Medal of Honor to Dr. Anita Newcomb McGee "in recognition of her splendid service to the United States in organizing a corps of trained nurses for the United States Army during the period of the Spanish-American War and the Philippine Insurrection." S. 2107, introduced by Senator Reynolds, North Carolina, proposes to authorize a pension of $50 a month to all persons who served seventy days or more in the military or naval service of the United States during the Spanish-American War who have reached the age of 65 years, including any woman who

served honorably as a nurse, chief nurse or superintendent of the Nurse Corps under contract. Contract surgeons of the Spanish-American War may not qualify for this pension if the bill is passed. S. 2134, introduced by Senator Schwellenbach, Washington, and H. R. 5727, introduced by Representative Coffee, Washington, propose to amend the Longshoremen's and Harbor Workers' Compensation Act to extend the benefits of it to boommen and raftmen employed in lumbering operations on navigable waters. S. 2138, introduced by Senator Capper, Kansas, proposes to establish in the United States Department of Agriculture a Division of Cooperatives to conduct research in cooperative problems and furnish advisory service to cooperatives. S. 2284, introduced by Senator Walsh, Massachusetts, proposes to authorize the President to appoint in the Navy 100 acting assistant surgeons for temporary service.

STATE MEDICAL LEGISLATION

California

Bill Introduced.—A. 2809 proposes that "All persons, having the ability to pay therefor, and the individuals authorized by the laws of this state to treat the physical and mental condition from which such persons are suffering, and for which hospital facilities are required, are entitled, without discrimination, to the full accommodations, advantages, facilities, services and privileges of any hospital" in the state.

Bills Amended in the Assembly.—A. 2764 was so amended April 28 as to make it the duty of any physician or other person attending a birth to treat both eyes of the infant within two hours after birth with a prophylactically efficient treatment. A. 2177, to amend the workmen's compensation act, was so amended April 25 as to permit chiropractors and osteopaths to render the "medical treatment" which must be furnished injured workmen.

Florida

Bills Introduced.—H. 392 proposes to repeal the present naturopathic practice act and to enact a new practice act for such practitioners. The bill proposes to define naturopathy as "that system or school of medicine which is taught and practiced in the standard schools and colleges of Naturopathy." Such licentiates apparently will have the right to use x-rays, electro-therapy, anesthesia, antiseptics, germicides, parasiticides, narcotics and antidotes. The bill states that "Physicians of the Naturopathic School of Medicine are to be of equal rank and grade as the physicians of the other schools of medicine designated as allopathic, homeopathic and eclectic, to have all rights, except to practice major surgery and to use materials not taught in the standard colleges or schools of Naturopathy." S. 263 proposes to make it the duty of any person attending a confinement case, within two hours after the birth of the child, to instil in each eye of the child a prophylactic solution prescribed by the State Board of Health for the prevention of ophthalmia neonatorum. S. 306 and H. 352 propose to establish at Avon Park, in Highlands County, a branch of the Florida State Hospital for the care of insane persons. H. 527 proposes, as a condition precedent to the issuance of a license to marry, that each party to the proposed marriage present a certificate from a licensed physician stating that the party has been given such examination, including a standard serologic test, as may be necessary for the discovery of syphilis, made not more than thirty days prior to the application, and that, in the opinion of the physician, the party is either not infected with syphilis or, if so infected, is not in a stage of that disease which is or may become communicable to the marital partner.

Illinois

Bills Introduced.—H. 876 proposes to enact a separate drugless therapy practice act and to establish a board of examiners to examine and license applicants for licenses to practice drugless therapy. The bill proposes to define drugless therapy as "a biological, physiological, chemical and material health science and as such employs physical, laboratory or other methods of diagnosis or analysis and such other procedures necessary to proper diagnosis and treat human ailments, diseases, defects, injuries, functions or dysfunctions, abilities or disabilities of the human body by or with manipulations or correction of any displaced tissue of any kind or nature and by or with the administration of, employment or performance of, diet, food, biological substances, vitamins, minerals, herbs or other substances normal to or necessary to the functions of the human body and including mechanotherapy, electrotherapy, obstetrics, phytotherapy, irradiation therapy, psychotherapy, hydrotherapy, thermotherapy, physiotherapy, and physical therapy." H. 922, to amend the motor traffic code, proposes that, on the trial of a defendant charged with driving while under the influence of intoxicating liquor, the court may admit evidence as to the amount of alcohol in the defendant's blood at the time of the alleged commission of the offense, as shown by a chemical analysis of his urine, saliva, breath or other body substance. Evidence that there was, at the time of the alleged commission of the offense, 5 hundredths of 1 per cent, or less, by weight of alcohol in the blood is prima facie evidence that the defendant was not under the influence of intoxicating liquor sufficiently to impair appreciably his driving ability. Evidence that there was at that time from 5 hundredths of 1 per cent to 15 hundredths of 1 per cent by weight of alcohol in the blood is to be relevant evidence but is not to be given prima facie effect in indicating that the defendant was under the influence of intoxicating liquor. Evidence that there was at that time 15 hundredths of 1 per cent or more by weight of alcohol in the blood is prima facie evidence that the defendant was under the influence of intoxicating liquor sufficiently to impair appreciably his driving ability. H. 978 proposes to grant to a hospital caring for a person injured through the negligence of another a lien on all rights of action, claims, judgments and compromises accruing to the injured person because of his injuries. The total amount of the hospital's lien, however, is not to exceed one third of the sum paid or due the injured person.

Massachusetts

Bill Introduced.—H. 2206 proposes to create a special committee to investigate the advisability of the commonwealth's furnishing oxygen tents to stated hospitals.

Michigan

Bills Introduced.—S. 419 proposes to enact a new workmen's compensation act and to provide compensation for workmen suffering disability because of injuries or diseases arising out of and in the course of employment. The employer must pay for such medical, surgical or other attendance or treatment, nurse and hospital service, medicine, crutches and apparatus for such period as the nature of injury or the process of recovery may require. An employee is to be permitted to select his own physician from a list compiled by the department of labor on the recommendation of the medical societies of the several counties. A. 785 proposes to enact a law to regulate the manufacture, distribution and advertising of foods, drugs, cosmetics and devices.

Missouri

Bill Introduced.—H. 717 proposes to condition the issuance of a license to marry on the presentation by both parties to the prospective marriage of certificates signed by the parties in the presence of and executed by a licensed physician or by the director of a laboratory approved by the Department of Health that the parties are free from all venereal diseases.

New York

Bill Introduced.—S. 2021 proposes to continue until March 15, 1940, the temporary state commission created by chapter 743, Laws, 1937, to examine, report on and recommend measures to improve facilities for care of hard of hearing and deaf children and children liable to become deaf.

Pennsylvania

Bills Introduced.—S. 545 proposes to prohibit the operation of a private nursing home or a private hospital without a license from the Department of Welfare. The present law requires licensure for private nursing homes or private hospitals operated for profit. H. 688 proposes to require all hospitals receiving appropriations from the commonwealth to pay each intern an annual salary of not less than $1,000.

H. 1409 proposes a procedure whereby hospitals may be reimbursed by the state for care and treatment rendered indigents injured in motor vehicle accidents. S. 677 proposes to authorize the board of commissioners of any county of the third class to enter into agreements with general hospitals within its boundaries for the care of indigents and part-pay patients afflicted with contagious diseases. S. 732 proposes to require every hospital receiving an appropriation from the state to conclude contracts with all nonprofit hospital service associations authorized to operate within its area. H. 1401 proposes to enact an occupational disease act to provide compensation to workers contracting occupational diseases, as defined in the act, in the course of their employment. The bill proposes to define "occupational disease" to mean only poisoning by arsenic, lead, mercury, manganese, phosphorus, methanol, carbon bisulfide, hydrocarbon distillates or halogenated hydrocarbons, benzol or nitro amido or amino derivatives of benzol; caisson disease; radium poisoning or disability due to radioactive properties of substances or to roentgen ray; poisoning by or ulceration from chromic acid or bichromate of ammonium, potassium or sodium; epitheliomatous cancer or ulceration due to tar, pitch, bitumen, mineral oil or paraffin; infection or inflammation of the skin due to oils, cutting compounds, lubricants, dust, liquids, fumes, gases or vapor; anthrax; silicosis or anthraco-silicosis; and asbestosis.

OFFICIAL NOTES

THE ST. LOUIS SESSION

Special Radio Programs

The following radio programs have been arranged in connection with the annual session of the American Medical Association in St. Louis:

Dr. Irvin Abell, President of the Association, will broadcast over the Blue network of the National Broadcasting Company Monday, May 15, from 4:15 to 4:30 p. m. central standard time. His subject will be "American Medicine Today." Dr. Rock Sleyster, President-Elect, will broadcast over the Columbia Broadcasting System Monday, May 15, from 3:45 to 4 p. m. Dr. Sleyster will be interviewed by Miss Jane Stafford of Science Service.

Dr. W. W. Bauer, Director of the Bureau of Health Education of the American Medical Association, will broadcast over the Blue network of the National Broadcasting Company Friday, May 19, from 4:15 to 4:30. He will present a summary of the scientific and sociological developments at the meeting.

RADIO BROADCASTS

The radio broadcasts by the American Medical Association and the National Broadcasting Company, under the title Your Health, continue as previously announced each Wednesday over the Blue network of the National Broadcasting Company at 1 p. m. eastern standard time (12 noon central standard time, 11 o'clock mountain time, 10 a. m. Pacific time).

Starting April 30, daylight saving time took effect in Chicago. The program is therefore being broadcast at the same hour Chicago daylight saving time, which means one hour earlier central standard time, two hours earlier mountain time and three hours earlier Pacific time. In communities where daylight saving time is in effect there is no change in the hour of the broadcast.

Owing to network conflicts the Chicago broadcast does not occur at 1 o'clock Wednesday but there is a rebroadcast from a recording over station WENR at 8 o'clock each Monday evening (8 o'clock Chicago daylight saving time, 7 o'clock central standard time). The program broadcast each Monday is identical with the network program of the preceding Wednesday.

The next three programs to be broadcast, together with their dates and their topics, are as follows:

May 17. Healthier Babies.
May 24. No broadcast because of radio conflicts occasioned by visit of British sovereigns to the United States.
May 31. Checking Up on Health.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

ARKANSAS

Dr. Vinsonhaler Resigns as Dean.—Dr. Frank Vinsonhaler, dean of the University of Arkansas School of Medicine, Little Rock, has announced his resignation, effective July 1. He will become dean emeritus.

Personal.—Dr. Oneal L. Atkinson has been appointed health officer of Hampton.——Dr. Ralph M. Sloan has been elected a director of the Jonesboro Chamber of Commerce.——Dr. Roy J. Turner has been appointed health officer of Fayetteville. ——Dr. Albert S. J. Clarke, formerly of Clarendon, has been transferred as health officer in charge of district number 16 at Ozark.

District Meetings.—The Fourth Councilor District Medical Society was addressed in Pine Bluff recently by Drs. William M. Adams and John Cash King, both of Memphis, Tenn., on "Acute Trauma of the Face" and "Cooperation Between the General Practitioner and the Roentgenologist." ——At a meeting of the Sixth Councilor District Medical Society in Hope April 6, the speakers included Drs. Thomas E. Strain, Shreveport, La., on "Hypoglycemia in Children"; Joseph E. Knighton Sr., Shreveport, "Chronic Duodenal Pathology."; Sidney J. Wolfermann, Fort Smith, "Socialized Medicine," and Thomas P. Foltz, Fort Smith, "Acute Traumatic Craniocerebral Injuries."——The Third Councilor District Medical Society was addressed at Forrest City April 13 by Drs. John C. Ayres, Memphis, Tenn., on "Anesthesia and Obstetrics"; Rudolph B. McCormick, Memphis, "Infectious Diarrheas in Children"; Sidney J. Wolfermann, "Duodenal Ulcer from the Medical and Surgical Standpoint," and William R. Brooksher, Fort Smith, "Compulsory Sickness Insurance."

CALIFORNIA

School Named in Honor of Dr. Widney.—The Crippled Children's High School, on the grounds of Polytechnic High School, Los Angeles, has been named the "Dr. Joseph Pomeroy Widney High School." The building was completed last year at a cost of $100,000. The late Dr. Widney was the founder of the old college of medicine of the University of Southern California and once served as dean and later president of the university.

COLORADO

Society News.—Dr. Jesse G. M. Bullowa, New York, discussed pneumonia before the Medical Society of the City and County of Denver April 4. Dr. Rudolf Schindler, Chicago, will discuss gastroscopy before the society June 6 and give a clinical demonstration.——At a meeting of the Northeast Colorado Medical Society in Sterling March 9, Dr. William R. Lipscomb, Denver, discussed "Hypertension and Its Surgical Treatment."——The Pueblo County Medical Society was addressed in Pueblo March 7 by Dr. Theodore E. Wade on "Industrial Minor Injuries." Dr. Guy H. Hopkins addressed the society recently on "Ocular Palsies." At the former meeting Dr. Frederic J. Peirce was elected to honorary membership.

CONNECTICUT

The Harvey Cushing Festschrift.—The March number of the *Acta chirurgica Scandinavica* is dedicated as a festschrift to Dr. Harvey Cushing, Sterling professor of neurology emeritus, Yale University School of Medicine, New Haven, and Moseley professor of surgery emeritus, Harvard Medical School, Boston. The issue contains twenty-five papers, all but two of which are in English.

Society News.—A symposium on cancer and its relation to public health was presented before the New Haven County Public Health Association March 2 by Dr. Matthew Griswold, chief of the cancer research division of the state department of health; Miss Elizabeth Fox, director, New Haven Visiting Nurse Association; Dr. Ashley W. Oughterson, director, tumor clinic, New Haven Hospital, and Dr. John Mendillo, director, tumor clinic, Grace Hospital.——Dr. Charles H. Best, Toronto, discussed "Heparin and Thrombosis" before the Yale Medical Society, New Haven, March 8.

State Medical Meeting in New Haven.—The one hundred and forty-seventh annual meeting of the Connecticut State Medical Society will be held at the Hotel Taft, New Haven, May 25-26, under the presidency of Dr. Hugh B. Campbell, Norwich. The speakers will include:

Dr. Roy D. McClure, Detroit, Modern Treatment of Burns—Reduction in Death Rate (movie).
Dr. Addison H. Bissell, Stamford, Treatment of Wounds.
Dr. Edward J. Ottenheimer, Willimantic, Factor of Delay in Recognition of Common Surgical Conditions.
Dr. James Leonard Vickers, Greenwich, Essentials in Fracture Treatment.
Dr. Claude C. Kelly, Hartford, Problem of Cleft Lip and Cleft Palate.
Dr. Chris H. Neuswanger, Waterbury, Urogenital Deformities.
Dr. Arthur S. Griswold, Bridgeport, Congenital Orthopedic Deformities.
Dr. William J. German, New Haven, Management of Congenital Defects of Nervous System.
Dr. Herbert Thoms, New Haven, Essentials of Pelvimetry (movie).
Dr. Carl H. Wies, New London, Rational Therapy.
Dr. Sidney S. Chipman, Norwalk, Vaccines of Value.
Dr. Berkley M. Parmelee, Bridgeport, X-Ray Fallacies.
Dr. Robert Glen Urquhart, Norwich, The Apical Cavity in Pulmonary Tuberculosis.
Dr. Francis G. Blake, New Haven, Sulfapyridine in the Treatment of Pneumonia.
Dr. James R. Miller, Hartford, Treatment Procedures for Cancer of the Uterus Advised by the Connecticut Tumor Clinics.
Dr. Carl E. Johnson, New Haven, Dysmenorrhea.

Among others who will discuss the papers will be Drs. Arthur W. Allen, Boston; Thomas H. Lanman, Boston; Chester S. Keefer, Boston, and John R. Fraser, Montreal.

DELAWARE

Dr. Morris to Direct Birth Control Federation.—Dr. Woodbridge E. Morris, Dover, director, division of maternal and child health and of the crippled children's division of the state board of health, has resigned to become director of the Birth Control Federation of America, with headquarters in New York. Dr. Morris graduated at Johns Hopkins University School of Medicine, Baltimore, in 1933. The federation was formed by the recent merger between the American Birth Control League and the Birth Control Clinical Research Bureau.

DISTRICT OF COLUMBIA

Cancer Campaign.—A cancer-control program was launched April 1. Speakers for lay audiences were provided by the speaker's bureau of the Medical Society of the District of Columbia. The campaign was to be concluded at the end of the month although the educational program is to be permanent.

Hospital News.—A department of syphilology will be established at Garfield Hospital to provide facilities for diagnosis, clinic treatment and hospitalization. To be known as department L, the new division will be in charge of Dr. William H. Hough, and Dr. Russell J. Fields will be director in charge of clinical work. There will also be a social service worker. Services and drugs will be free to those unable to pay.

Society News.—At a meeting of the Washington Ophthalmological Society March 6 Drs. Walter O. Teichmann and Edgar Leonard Goodman spoke on "Skin Lesions Around the Eye" and "Muscle Tendon Transplantation for Paralysis of External Rectus."——The George Martin Kober Medical Society was addressed recently by Dr. Frank M. Hand on "A General Reference to the Extremities of the Body" and Otto H. Wendt, D.D.S., "Eruption of Three Sets of Superior Central Incisor Teeth: Case Report."——Dr. William F. Rienhoff Jr., Baltimore, discussed "Peptic Ulcer" before a meeting of the naval medical and dental officers March 6.——A panel discussion on "Recent Advances in the Anemias" was presented before the Washington Society of Pathologists May 10; Dr. Elbert DeCoursey was the mediator and Dr. Roger M. Choisser the leader.

GEORGIA

Society News.—The Ware County Medical Society was addressed in Waycross April 5 by Dr. Thomas J. Ferrell, Waycross, on "Ruptured Peptic Ulcer."——At a meeting of the Colquitt County Medical Society in Moultrie March 17 Dr. James A. Redfearn, Albany, spoke on "The Nervous Patient."——A meeting of the Georgia Medical Society in Savannah March 28 was addressed by Dr. Richard F. Slaughter, Augusta, on "Cranial Cerebral Trauma." Dr. Samuel F. Rosen, Savannah, reported a case of Bazin's disease.——At a meeting of the Fulton County Medical Society April 20, among others, Drs. William Howard and Hugh E. Hailey discussed "Pruritus Ani et Vulvae and Its Relation to Other Allergic Conditions." Among others, Dr. John F. Denton, Atlanta, presented a paper before the society April 6 entitled "Radium and X-Ray in the Treatment of Gynecologic Conditions."

IDAHO

Changes in Health Officers.—Dr. George H. Bischoff, health officer of Bannock County, has been appointed director of the maternal and child welfare and crippled children's division of the state department of health. He will be succeeded by Dr. James V. Foley.

Society News.—A recent meeting of the North Idaho Medical Society was addressed in Lewiston by Drs. Jean D. Kindschi and Robert H. Southcombe, both of Spokane, Wash., on "Syphilis in Pregnancy" and "Newer Treatments in Psychiatry" respectively.——A special meeting of the Pocatello Medical Society was recently addressed by Drs. Cleveland J. White on skin diseases; James K. Stack, injuries of the wrist, and Stanley Gibson, treatment of respiratory diseases in children. The same speakers who are members of the staff of Northwestern University Medical School, Chicago, addressed the South Side Medical Society recently and the Southwestern Idaho District Medical Society.

ILLINOIS

Physician Honored.—Dr. William F. Scott, Maywood, for many years chief of staff of the Oak Park Hospital, was guest of honor at a dinner April 26 given by the Sisters of Misericorde in honor of his many years of service as chief of the staff of the hospital. Members of the staff presented to the hospital an oil portrait of Dr. Scott, which is to be hung in the hospital gallery, and Dr. Scott was presented with a memorial volume containing the signatures of his friends and associates. Dr. Austin A. Hayden, Chicago, was toastmaster.

Chicago

Dr. Szent-Gyorgyi Will Speak on Oxidation.—Dr. Albert Szent-Gyorgyi, professor of medical chemistry, Royal Hungarian Franz Josef University, Szeged, Hungary, will lecture in the Palmer House May 26 under the auspices of the Institute of Medicine of Chicago, on "The Mechanism of Biological Oxidation."

The Annual Hedblom Lecture.—Dr. John Alexander, professor of surgery, University of Michigan School of Medicine, Ann Arbor, will deliver the annual Hedblom lecture sponsored by the Phi Beta Pi Fraternity of the University of Illinois College of Medicine May 17. His subject will be "The Management of Bronchiectasis and Abscess of the Lung."

Personal.—Dr. Max Cutler, director of the Chicago Tumor Institute, recently returned from a cruise around South America in which he gave nine addresses before medical associations and universities. The Chicago Tumor Institute has granted one fellowship to each of the South American countries for graduate instruction in the field of cancer. The fellows will be selected by the deans of the medical schools and the ministers of health.

INDIANA

Society News.—Dr. James O. Ritchey discussed "Serum Treatment of Pneumonia" before the Indianapolis Medical Society April 25 and Drs. Kenneth G. Kohlstaedt and Irvine H. Page, "Sulfapyridine Treatment of Pneumonia." Dr. Andrew C. Ivy, Chicago, discussed "Practical Application of the Newer Concepts of the Physiology of the Digestive System" before the society recently. A symposium on gynecologic surgery was presented before the society March 28 by Drs. Carl Habich, Dudley A. Pfaff, John William Hofmann and Ross C. Ottinger.

IOWA

Instructors in First Aid.—Forty-nine persons received first aid proficiency certificates at the graduation exercises of a Red Cross first aid training course recently completed in Fremont County. Twelve physicians of the county medical society, under the direction of the president, Dr. Ralph Lovelady, Sidney, served as instructors for the group of sixty-two adults, who met each week from Oct. 31, 1938, to Feb. 6, 1939. Personal instruction in bandaging and splinting procedures was given and a demonstration of various resuscitation appliances was conducted. In addition, five minute talks on symptoms, home treatment and prevention of some of the communicable diseases were presented.

KENTUCKY

Home of Ephraim McDowell Restored.—The home of Dr. Ephraim McDowell at Danville has been restored as a memorial to him and to his patient, Mrs. Jane Todd Crawford, and will be formally dedicated May 20. The dedicatory address will be delivered by Dr. Irvin Abell, chairman of the memorial

committee, and President, American Medical Association, and Gov. A. B. Chandler will give the speech of acceptance. A dinner will be held in the evening. The restoration work was done by the WPA and the Commonwealth of Kentucky. The home was presented to the state by the Kentucky State Medical Association.

MAINE

Society News.—Dr. Thomas A. Foster, Portland, discussed "Pyuria in Infants and Children" before the Androscoggin County Medical Association, Auburn, March 16.——Dr. Richard H. Overholt, Brookline, discussed "Developments in the Management of Chest Lesions" before the Cumberland County Medical Society April 21 in Portland.——Dr. Samuel A. Levine, Boston, addressed the Kennebec County Medical Association, Gardiner, April 20, on "Auscultation of the Heart."——At a meeting of the Knox County Medical Association in Rockland, April 11, Dr. Siegfried J. Thannhauser, Boston, spoke on vitamin deficiencies.

MASSACHUSETTS

Health Week in Lynn.—A special program sponsored by the Lynn Young Men's Christian Association marked the twelfth annual "Greater Lynn Health Week" March 11-19. "Youth Health" was the theme of this year's observance. Lectures, radio broadcasts and exhibits were features of the program.

Society News.—Dr. Harry Goldblatt, Cleveland, discussed "Experimental Observations on the Pathogenesis and Treatment of Hypertension" before the New England Heart Association April 28.——Among others, Drs. Frank H. Lahey and Everett D. Kiefer, Boston, addressed the New England Roentgen Ray Society April 21 on "Carcinoma of the Thyroid" and "Lesions of the Small Bowel."——The speakers before the New England Pathological Society in Wrentham April 20 included Drs. Clemens E. Benda, Wrentham, on "The Bone Development in Mongoloid Deficiency"; Thomas G. Cogswell, Worcester, "Epidermoid Cysts"; Harry M. Zimmerman, New Haven, Conn., "Nervous System Lesions in Hyperinsulinism," and Milton C. Winternitz, New Haven, on "Arteriosclerosis."

MICHIGAN

The Hickey Lecture.—Dr. Byrl R. Kirklin, Rochester, Minn., delivered the third annual Hickey Memorial Lecture before the Wayne County Medical Society, Detroit, April 3 on "Bleeding Lesions of the Gastro-Intestinal Tract and Their Roentgenologic Diagnosis."

Portrait of Dr. McKean.—An oil painting by Roy Gamble of the late Dr. George E. McKean will, when completed, be placed permanently in the headquarters of the Wayne County Medical Society. It will be unveiled at the annual meeting of the county medical society. Dr. McKean was president of the society 1919-1920 and served on the board of trustees from 1921 to 1933.

Examination of Mexican Beet Field Workers.—A cooperative plan for protecting the health of Michigan communities from contagious diseases among imported Mexican beet field workers was announced March 10 by the state department of health. The four state associations of sugar beet growers have agreed to pay half the expense of health examinations of field workers before they are brought from Texas and to employ no worker who has not passed a physical examination showing that he is free from tuberculosis and syphilis in infectious stages. The state health department will provide the other half of the cost of the examinations with the aid of funds allotted from the U. S. Public Health Service. The examinations will be made in Texas before the workers are hired by the sugar beet growers' associations. About 10,000 Mexican beet field workers are imported into the state during each growing season and diseases found among these laborers in the past have been a danger to the health of several Michigan communities. According to the state department, an outbreak of Shiga dysentery in Shiawassee County last summer was attributed to this source.

MISSISSIPPI

Personal.—The Gulf Coast Medical Society recently gave a dinner in honor of Dr. Daniel J. Williams, Gulfport, who has been health officer of Harrison County for twenty-five years; he is also a past president of the state medical association.——Dr. William W. Reynolds, Meridian, was recently appointed superintendent of the Matty Hersee Hospital, a state-supported institution, succeeding Dr. George Lamar Arrington.

Society News.—The Delta Medical Society was addressed in Greenville April 12 by Drs. Ira B. Bright, Greenwood, on "Electrocardiogram as an Aid to Diagnosing Heart Conditions"; Joseph G. Peeler, Shaw, "Sulfanilamide—Its Use in General Practice"; Onnie P. Myers, Indianola, "The Management of Diabetes in Country Practice"; Earl T. White, Greenville, "Cancer of the Colon"; James R. Jackson, Belzoni, "Allergy," and Orren W. Hyman, Ph.D., Memphis, Tenn., "What the Present Trend Toward Socialized Medicine Is Going to Do to the General Practitioner."

MONTANA

A Pneumonia School.—According to the *Journal-Lancet*, a pneumonia school available to physicians and laboratory technicians in western Montana was recently held at Montana State University, Missoula, under the auspices of the university and the state board of health. The speakers were Drs. Ernest D. Hitchcock, Great Falls; Meredith B. Hesdorffer, Missoula; Raymond F. Peterson, Butte; Burton K. Kilbourne, Helena, and Miss Edith Kuhns, Helena, director of the hygienic laboratory for the state board of health.

NEW JERSEY

New Committee to Control Cancer.—A New Jersey State Committee for the Control of Cancer has been organized to coordinate activities of lay and professional groups, under the auspices of the county medical societies and the American Society for the Control of Cancer. The committee aims to assist medical societies in securing facilities for the treatment of cancer as well as to carry on a campaign of educational publicity. Dr. Joseph H. Kler, New Brunswick, chairman for New Jersey of the American Society for the Control of Cancer, is also chairman of the new committee which is composed of representatives of both medical and non-medical groups.

NEW YORK

Smallpox Reported.—Smallpox has occurred in the village of Colonie, Albany County. The first case was in a man, never vaccinated, who conducts a gas station and trailer camp at stop 19 on the Albany-Schenectady Road. A systematic follow-up of all persons known to have been exposed to the gas station proprietor and his wife was carried out. Widespread publicity was given to the occurrence of these cases.

Personal.—Dr. George Emory Lochner, Albany, was guest of honor at a dinner March 15 given by the board of trustees of the Anthony N. Brady Maternity Home to celebrate his completion of fifty years in the practice of medicine. Dr. Lochner has been chief of staff of the Brady home since its establishment.——The Dutchess County Medical Society gave a dinner April 12 in Poughkeepsie in honor of Dr. Robert W. Andrews, who is moving to Texas after practicing in Poughkeepsie more than forty years. Dr. John C. McClintock, Albany, addressed the meeting of the society on thyroid problems.

New York City

Committee to Study Forensic Psychiatry.—The mayor has appointed a group of physicians and lawyers to serve as a committee to originate a plan of forensic psychiatry to pass on the mentality of persons under charges in New York courts, it is reported. The appointment was made when provision for pay for lunacy commissions was dropped in the city budget. The New York Academy of Medicine, the Bar Association of the City of New York and the New York County Lawyers Association will cooperate in the study which will be in charge of William B. Herlands, commissioner of investigation. Included on the committee will be Drs. John A. Hartwell and Israel Strauss, Mr. George Sylvester and Mr. Irving Brawer.

Another Physicians' Orchestra.—Announcement is made of the formation of the Doctors Orchestral Society of New York. Officers are Drs. Leopold Glushak, president; William S. Thomas, vice president; Charles Auer, treasurer, and Harold Belcher, secretary. Ignatz Waghalter is the conductor. The society plans to hold its premiere in the Town Hall May 26 with Ernest Schelling, American pianist-composer, as soloist. The proceeds of the affair will be donated to the Physicians' Home and the Loan and Relief Fund of the Medical Society of New York. The program will include:

Overture to "Oberon"	Weber
Symphony No. 5 in E minor "From the New World"	Dvořák
Concerto for Piano and Orchestra in A minor	Schumann
"Emperor" Waltz	Strauss

Personal.—Dr. Chrisman G. Scherf, medical superintendent of Metropolitan Hospital, has been named to a similar position at the new Hospital for Chronic Diseases on Welfare Island, according to a recent announcement by the commissioner of hospitals, Dr. Sigismund S. Goldwater. The new hospital will open about July 1, it was stated.——Dr. Victor G. Heiser received the gold medal of the Holland Society of New York April 6 in recognition of his "distinguished service to public health."——Dr. Alexis Carrel of the Rockefeller Institute for Medical Research was awarded the gold service medal of the Rotary Club of New York April 13 "in recognition of a life devoted to the amelioration of human suffering."——Dr. Shirley W. Wynne, former health commissioner, has been named commissioner of the Manhattan Council, Boy Scouts of America. ——Dr. Thomas M. Rivers, director of the Hospital of the Rockefeller Institute for Medical Research, has been appointed to the New York City Board of Health by Mayor La Guardia to succeed the late Dr. Carl Boettiger.

NORTH CAROLINA

District Meetings.—Dr. Beverley R. Tucker, Richmond, Va., was the guest speaker at a meeting of the Second District Medical Society in Greenville April 13, on advancements in the treatment of the mentally ill.——The Fifth District Medical Society held a meeting in Lumberton April 6 with the following speakers, among others: Drs. David Henry Poer, Atlanta, on surgical management of goiter; James M. Ruffin, Durham, diagnosis and treatment of conditions causing bloody diarrhea, and Russell S. Beam, Lumberton, sulfapyridine in pneumococcic infections.——The Tenth District Medical Society met in Asheville March 31 in conjunction with the state board of health and the extension bureau of the University of North Carolina. Dr. Robert E. Seibels, Columbia, S. C., spoke on the care of pregnant women. Dr. Horton R. Casparis, Nashville, Tenn., spoke on care of children in infancy.

OHIO

Personal.—The John and Mary Markle Foundation, New York, has awarded a grant of $4,100 to Dr. Carl J. Wiggers, professor of physiology, Western Reserve University School of Medicine, Cleveland, to study the nature of ventricular fibrillation.——Dr. Harry Goldblatt, Cleveland, recently received the "grand scientific award" of the Phi Lambda Kappa fraternity in recognition of his work on essential hypertension.

The Rachford Lectures.—Dr. Ernest W. Goodpasture, professor of pathology, Vanderbilt University School of Medicine, Nashville, Tenn., delivered the Benjamin Knox Rachford Lectures at the University of Cincinnati April 13-14. His subjects were "Experimental Virus Infections of the Chick Embryo" and "Experimental Bacterial Infections of the Chick Embryo." The Rachford Lectureship was established in 1929 in memory of Dr. Rachford, who organized the department of pediatrics in the university.

PENNSYLVANIA

Narcotic Addicts and Epileptics Barred from Driving.—More than 28,000 known narcotic addicts have been barred from obtaining licenses and driving permits in Pennsylvania. They will not be allowed to drive until they can show on competent medical authority that they are cured. The report also said that 5,000 applicants known to be subject to epilepsy have been refused permits and 259 are waiting for special hearings.

Philadelphia

Sectional Meeting on Physical Therapy.—The spring session of the American Congress of Physical Therapy, under the auspices of the New York Physical Therapy Society and the Pennsylvania Physical Therapy Association, was held at Jefferson Medical College, Philadelphia, April 22. Among the speakers were:

Dr. John H. Willard, Abdominal Neuralgia.
Dr. Karl Harpuder, New York, Physical Therapy in Peripheral Vascular Disease.
Dr. Louis Kaplan, Physical Therapy in Fractures Treated with Unpadded Cast.
Dr. Kristian G. Hansson, New York, After-Treatment of Infantile Paralysis.
Dr. Abraham R. Hollender, Chicago, Controversial Problems in Nasal Iontophoresis.
Dr. Gustave Bucky, New York, New Developments in Grenz Rays.
Dr. Jean Saidman, director, Institute of Actinology, Paris, New Methods of Physical Diagnosis and Physical Therapy.
Dr. Joseph T. Beardwood Jr., Philadelphia, Newer Methods of Physical Therapy in the Treatment of Complications of Diabetes.

Pittsburgh

Second Renziehausen Lecture.—Frank G. Young, Ph.D., a member of the scientific research staff of the National Institute for Medical Research, London, England, delivered the second Renziehausen Memorial Lecture at the Mellon Institute April 24. His subject was "The Anterior Pituitary Gland Diabetes." These lectures were established recently by Miss Emilie Renziehausen in memory of her brothers.

SOUTH CAROLINA

Hospital News.—A new Oconee County hospital at Seneca was opened February 6 with accommodations for forty patients. Construction of the hospital was a WPA project representing an outlay of about $75,000.

Society News.—Dr. Frederic W. Schlutz, Chicago, addressed the Columbia Medical Society in Columbia April 10 on "Sulfanilamide Therapy in Pediatric Practice" and Dr. LeGrand Guerry, Columbia, "Reconstruction of Bile Passages Based on a Study of Fifteen Consecutive Cases."

VIRGINIA

Personal.—William T. Sanger, LL.D., president of the Medical College of Virginia, Richmond, received the honorary degree of doctor of humane letters, from Bridgewater College April 3, when he made a Founder's Day address. Dr. Sanger will receive the degree of doctor of laws from the University of Richmond at its June commencement.——Dr. James Brooke Pettis, recently on the staff of the New Jersey State Hospital, Marlboro, N. J., has been appointed director of clinical psychiatry.

Society News.—Dr. Tiffany J. Williams, Charlottesville, addressed the Danville-Pittsylvania Academy of Medicine, April 11, on "Toxemias of Pregnancy."——Dr. Hugh C. Henry, Richmond, has been elected president of the Neuropsychiatric Society of Virginia; Dr. Thomas N. Spessard, Norfolk, vice president, and Dr. Edward H. Williams, Richmond, secretary.——Dr. Edwin C. Hamblen, Durham, N. C., was the guest speaker at the spring meeting of the Southwestern Virginia Medical Society in Bristol April 13 on endocrinology.

WASHINGTON

Hospital News.—A new Shriners' Hospital was dedicated recently in Spokane adjacent to St. Luke's Hospital, where the original building was erected in 1924. Erected at a cost of $85,000, the new building will provide facilities for the care of crippled children.

Spokane Surgical Meeting.—Dr. Edward William Alton Ochsner, New Orleans, was the guest speaker at the annual meeting of the Spokane Surgical Society April 8. Dr. Ochsner gave the following addresses: "Peripheral Vascular Disease," "Thrombophlebitis" and "Surgical Significance of Amebiasis." Members of the society presented papers and conducted clinics.

Graduate Course.—The University of Washington announces its annual extension graduate medical course July 17-21. Instructors this year will include Drs. Andrew C. Ivy, Howard C. Ballenger, Newell C. Gilbert, George H. Gardner and Charles Marshall Davison, all of the staff of Northwestern University Medical School, Chicago. Clinics will be conducted at King County Hospital by local physicians.

WEST VIRGINIA

Personal.—Dr. Walter E. Vest, Huntington, will receive the honorary degree of doctor of science at the June commencement of the Medical College of Virginia, Richmond. Dr. Vest graduated from the college in 1909.

Society News.—Dr. Samuel Iglauer, Cincinnati, addressed the Ohio County Medical Society, Wheeling, April 21, on "Deep Infections of the Neck and Mediastinum."——Dr. Thomas E. Jones, Cleveland, addressed the Ohio County Medical Society April 7 on "Abdominoperineal Resection" and Dr. Leon Herman, Philadelphia, March 24, on "Urinary Infections in the Pregnant Woman."——Dr. Hugo Roesler, Philadelphia, addressed the Kanawha Medical Society, Charleston, April 11 on "Nonmechanical Diagnosis of Heart Lesions."

WISCONSIN

Society News.—Dr. John W. Harris, Madison, addressed the Milwaukee Society of Clinical Surgery March 28 on "An Evaluation of the Various Operative Procedures for the Correction of Uterine Prolapse" and Dr. Edmund H. Mensing,

Milwaukee, on "Prophylaxis of Peritonitis."——The Medical Society of Milwaukee County and the Milwaukee County Dental Society held a joint meeting March 10; the speakers were Dr. Elmer L. Sevringhaus, Madison, on "The General Features of Mineral Metabolism" and Isaac Schour, Ph.D., D.D.S., Chicago, "Calcification of the Tooth as an Index of the Constitutional Pattern of the Child."

GENERAL

American Board Examinations in Ophthalmology.—The American Board of Ophthalmology will conduct a written examination in various cities of the United States, in Honolulu, Puerto Rico and Canada August 5. Formal application for this examination must be received before July 1. General oral examination for successful candidates will be held in Chicago October 7. For information, write at once to the secretary, Dr. John Green, 6830 Waterman Avenue, St. Louis.

Awards for Chemical Research.—Among awards for research in chemistry for the academic year 1939-1940 by the Lalor Foundation, Wilmington, Del., are the following of medical significance: Otto Karl Behrens, Ph.D., Rockefeller Institute for Medical Research, New York, for work on chemistry of peptide metabolism; Andrew Calvin Bratton, Ph.D., instructor in pharmacology and experimental therapeutics, Johns Hopkins University School of Medicine, Baltimore, chemical aspects of chemotherapy of compounds of the sulfanilamide type.

Health Conference in Rio de Janeiro.—The eighth biennial conference of the Health Section of the World Federation of Education Associations will be held in connection with the congress of the federation in Rio de Janeiro, Brazil, August 6-11. A working conference is being developed in which all present will share experiences in health education, health services and physical education in the schools. Brief outlines of each paper will be read when the author, or a representative of his country, is present and before the close of each of the four sessions a summary of the papers and discussions will be presented and acted on by the group. Program and travel information can be obtained from the executive secretary, Health Section Secretariat, 200 Fifth Avenue, New York.

Association for the Study of Goiter.—The American Association for the Study of Goiter will hold its annual meeting in Cincinnati May 22-24. The tentative program includes the following speakers:

Dr. Willard O. Thompson, Chicago, Management of Severe Thyrotoxicosis.
Dr. Elmer C. Bartels, Boston, Coronary Disease in Myxedema.
Dr. Douglas U. McGregor, Hamilton, Ont., Tuberculosis of the Thyroid Gland.
Drs. Samuel F. Haines, Thomas B. Magath and Fred H. Powers, Rochester, Minn., The Hippuric Acid Test in Hyperthyroidism.
Drs. Brien T. King and Frederick Lemere, Seattle, A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis.
Dr. Warren H. Cole, Chicago, Anesthesia in Thyroidectomy with Special Reference to Avertin.
Drs. Elliott C. Cutler and Stanley O. Hoerr, Boston, Avertin Anesthesia for Thyrotoxicosis.

Awards in National Traffic Safety Contest.—New Jersey won the grand prize for states and Providence, R. I., the grand prize for cities in the annual national traffic safety contest sponsored by the National Safety Council. All states and 1,163 cities participated in the contest. Three other states won first place in their geographic divisions: Oklahoma, Iowa and Washington. Six cities won awards in their population groups: Cleveland and Milwaukee tied among cities with populations of 500,000 or more; Trenton, N. J., 100,000 to 250,000; Saginaw, Mich., 50,000 to 100,000; Waukegan, Ill., 25,000 to 50,000, and Mason City, Iowa, 10,000 to 25,000. Providence won first place in the 250,000-500,000 population group. In addition to these awards the judges gave prizes and honorable mention and named a special honor roll of 157 cities with populations between 5,000 and 10,000 which went through 1938 without a traffic death. New Jersey cut its traffic deaths in 1938 to 885 from 1,378 in 1937. Providence had sixteen traffic deaths in 1938 as compared with forty-one in 1937. The average death rate for all prize winning cities in the contest was 9.9 per hundred million miles traveled; the national average for cities was 14.5. Scoring of the contest was based on a maximum score of 100 possible points. The motor vehicle death rate counted for 50 points and the remaining fifty points were divided among accident reporting systems, traffic engineering and enforcement, child safety programs and public education. Bronze plaques were presented to the governors of the states and mayors of the cities were presented at a dinner at the Mayflower Hotel, Washington, D. C., April 13.

Foreign Letters

LONDON

(From Our Regular Correspondent)

April 15, 1939.

Sulfapyridine in the Treatment of Pneumonia

The use of sulfapyridine for pneumonia is the leading therapeutic topic of the day. At the Royal Society of Medicine Dr. L. E. H. Whitby said that the efficacy of this drug had been fully confirmed. Provided the patient could live for twenty-four hours and retain some 5 Gm. the chance of success was greatly increased, whatever the day of the disease on which the treatment was begun. The scheme of dosage of Gaisford and Evans, from 4 to 5 Gm. the first twenty-four hours and 3 Gm. daily thereafter to a total of 25 Gm., was the usual one followed. Stopping administration of the drug within four days was followed by an exacerbation, which in animals was difficult to control. Whether passive immunization with serum or active immunization with vaccine should be combined with the use of sulfapyridine was difficult to say. Patients treated on the fifth day and afterward required only 15 Gm. to produce a crisis and cure. The basic indication for the drug was a definite coccic infection. It had no action on influenza. Its chief disadvantage was gastric irritation and vomiting in about 30 per cent of cases.

Dr. W. F. Gaisford reported a year's hospital experience with sulfapyridine in which he had treated between 600 and 700 patients suffering from infections of the respiratory tract. Of these 400 had lobar pneumonia. Children under the age of 5 years and patients dying within twelve hours of admission to the hospital were not included in the statistics. The total mortality dropped from 23 per cent before the use of the drug to between 6 and 7 per cent. In patients over 50 the drop was from 50 to 24 per cent; in patients under 50, from 17 to 1.6 per cent. In nearly all cases the effect on the temperature was shown by a fall to normal within forty-eight hours of the beginning of treatment. Continuance of pyrexia indicated either a complication or an organism that was not the pneumococcus. There was no indication of any alteration in the rate of resolution. The general condition rapidly improved after the fall in temperature. Toxic effects of the drug were slight, rashes similar to but less severe than those produced by sulfanilamide. Cyanosis was observed in some cases, but it ceased to be a worrying sign. Vomiting sometimes occurred but was rarely so persistent as to prevent retention of a sufficient dose of the drug.

Prof. Alexander Fleming said that sulfapyridine did not seem to kill the pneumococcus, which presumably was killed by the natural defenses of the body. The mortality might be still further reduced if patients were immunized when the drug was given. There was not much time to develop active immunity, but animal experiments indicated that not much active immunity was required when combined with drug treatment.

Dr. Maurice Davidson sounded a note of warning against the indiscriminate way the sulfanilamides were being used by the junior members of the profession. We were not yet in a position to say that they did not cause damage. A drug so potent as to be able to deal effectively with a virulent organism might possibly damage renal epithelium or even the myocardium. The president, Dr. H. L. Tidy, said that the responsibility for the wild use of these drugs in general practice lay with the travelers of drug firms.

Operation for Paralyzed Muscles

The physiologist Prof. W. A. Osborne of Melbourne, in conjunction with Basil Kilvington, showed in 1907 that the regenerating fibers of the cut sciatic nerve of the dog do not travel down the original paths but even cross from one division

to another. The Australian surgeon N. D. Royle has ingeniously turned this pathologic fact to account in the treatment of paralysis (*M. J. Australia* **1:227** [Feb. 11] 1939). He points out that in anterior poliomyelitis degeneration of a peripheral nerve may follow degeneration of the spinal neuron and that the nerve trunk may contain both normal fibers and sheaths with degenerated fibers. If the nerve is interrupted and allowed to regrow, some healthy fibers will grow into sheaths containing degenerated fibers and so reach the paralyzed muscles, which will regenerate, for Royle has obtained return of power in four successive cases. In one the tibialis anticus was paralyzed and in another all the muscles supplied by the median and ulnar nerves. In the latter case nerve suture and transplantation were performed, in addition to nerve section below the line of transplantation. Royle now adds another typical application.

A boy aged 4 had paralysis from injury of the brachial plexus at birth. There was loss of power in the hypothenar eminence and in the interosseus muscles of the left hand, but the muscles of the forearm supplied by the ulnar nerve had recovered. Royle exposed the ulnar nerve above the elbow and crushed it with strong artery forceps in several diameters so as to disorganize the axis cylinders. A year later the boy had regained power in the paralyzed muscles. The muscles considered by Royle suitable for this operation are the tibialis anticus, which in poliomyelitis often remains paralyzed when other muscles of the anterior tibial group recover, the intrinsic muscles of the thumb, the interossei, the peronei, individual muscles of the posterior peroneal group and any paralyzed muscle in an otherwise normal group. The crushing of the nerve must be done at some distance proximal to its first muscle supply. Thus in the common peroneal nerve it must be done at least 5 cm. above the origin of the supply of the muscles and in the ulnar nerve 1 inch or more above the elbow. The usual orthopedic practice is followed while recovery of the paralyzed muscles is being awaited.

PARIS

(From Our Regular Correspondent)

April 8, 1939.

Prevalence of Brucellosis in France

A report was presented at the February 21 meeting of the Académie de médecine of Paris by Drs. Dubois and Sollier on the incidence and prevention of brucellosis in France. More than 4,000 cases were reported last year, with a mortality of from 6 to 7 per cent. The disease is almost exclusively of animal origin. Of 100 cases, the infection originates in about eighty in sheep, in fifteen in goats and in only about five in cows. There is danger, however, of pigs acting as sources of infection. With the exception of certain portions of Brittany the disease has been reported from every department in France. The object of the report was to draw attention to the inefficacy of present sanitary laws against the disease and the necessity of vaccination of every person in an infected area. Experience has shown that only about one case in twenty or twenty-five is reported. Fortunately this does not have any serious results, because disinfection of the surroundings of the patients with brucellosis has been shown to be of little value. Interhuman contagion is exceptional. The chief value of a declaration of the disease is that it enables the authorities to discover new foci of animal brucellosis and thus enables human beings in that locality to be aware of the dangers surrounding them. Unfortunately, owing to the small number of cases reported by farmers, most of the centers of infection in sheep and goats remain undiscovered. The majority of the sanitary measures are inadequate because of certain circumstances, which include the fact that the mode of contagion is complex in animals; there are certain species which are specially susceptible and there is a possibility for the female to be apparently well and yet be a carrier to an unlimited degree.

VACCINATION

As to prophylaxis, efforts to vaccinate animals against the disease have given only negative results. In general only killed bacteria have been used, but Dr. Dubois has employed since 1934 living and virulent cultures of Brucella abortus suis. Sheep vaccinated by this method have resisted experimental brucellosis as well as a natural form of the disease. At the same time they have not been infectious for animals of the same species in spite of a contact of from one to two years. A study of 480 cases of undulant fever since 1930 shows that seven of eight persons were infected after direct or indirect contact with infected animals and only one of eight after ingestion of milk or cheese. It is impossible to avoid infection of persons whose daily work brings them into constant contact with infected animals. The only method of prophylaxis for human beings is the use of a polyvalent vaccine containing strains of Brucella melitensis of human, sheep and goat origin and also strains of Brucella abortus bovis and suis. The use of such vaccine was begun by the authors in 1929, and more than 5,000 persons have been vaccinated, of whom 3,800 were farmers and 1,200 persons engaged in slaughterhouses or veterinary work. The unquestionable value of this method of vaccination is shown by the fact that undulant fever was observed in numerous nonvaccinated persons, whereas those who had been equally exposed but previously vaccinated were immune.

BERLIN

(From Our Regular Correspondent)

March 27, 1939.

The New Wiener Medizinische Gesellschaft

In the train of events under the new régime in Austria the famous Gesellschaft der Aerzte, like many other time-honored organizations, has lately been dissolved. In 1937 the centenary of the society was celebrated in a manner befitting so illustrious a medical body. The greatest German names in the world of medicine enthusiastically participated in this celebration. The list of speakers and scientific lecturers was brilliant and in keeping with the accomplishments of the society and its reputation. But its continuance was not to be tolerated by the Nazi rulers of Vienna, and, like all other medical societies, it was disestablished, in the 101st year of its existence. In its stead a new Wiener medizinische Gesellschaft was created, designed to serve medical practitioners and scientific research. Naturally only Aryan doctors are admitted to membership and in addition foreigners who are unquestionably in sympathy with the régime. The plan of the new society includes sections for the various special disciplines to take the place of the former societies of specialists. There is also newly added a special section for military medicine. The president of the new society is no distinguished clinician; he is the Nazi district governor of Vienna, that is to say a politician who is also an official of the Nazi bureau of national health.

In his inaugural address, February 4, the new president himself stated that he was well aware that many representatives of science were not wholly in accord with the establishment of the new organization under political rather than under scientific auspices. The president is Dr. O. Planner-Plan. This political control has become necessary, he stated, because as district governor he is better informed about local public health affairs than any one else. Since he has the representatives of science at his beck and call, it is possible for him to draw on consultant opinion in any problem which may arise. As he put it, "Prominent representatives of the various specialties may be commissioned to give an opinion with regard to any questions, and thus their abundant knowledge will be made to serve the nation's health." Dr. Planner-Plan's address was embroidered with a wealth of detail, which may be briefly summarized: The present speedy tempo of the national work program in Germany

not only should be maintained but should be surpassed. This means that a maximal expenditure of energy will be demanded of all German workers and soldiers. A further objective is conservation of the prolonged efficiency of the nation's workers. In this connection the physicians have an important mission to fulfil. Specializing physicians, too, have their respective duties, which the president went on to enumerate.

At each session of the new society a lecture and demonstrations will be given. It is further planned to correct certain bad features of medical practice; above all, an endeavor will be made to effect better collaboration between directors of clinics and specialists active as consultants.

THE NAZI CREED WITH REGARD TO MEDICINE

The principal address at the inaugural session was delivered by Prof. Franz Hamburger, ordinarius in pediatrics (and successor of Pirquet), long known for his Nazi sympathies. He climaxed his talk with the following pronouncement: "National socialism means a revolution in every sphere of our civilization and culture. No phase of western culture is unaffected by it. Most noteworthy of all, and what must remain most noteworthy, is the revolution in the realm of medical science, in the field of public health." The speaker went on to say that, despite the achievements of natural science within the past 150 years, medicine had been on the wrong track. "Medicine has now progressed beyond its old frontiers and has broken out of its shell, thanks to the philosophy and deeds of the führer." The healing art of yesterday has become the planned hygiene of today, the medical knowledge of mankind. Thus pediatrics becomes the medical study of children, gynecology the medical study of women, and so on. "With admirable clarity and logic the führer points the way into these fields, "like a physician by the grace of God he shows us the path to health." The rubbish of which physicians must free themselves is the dross of misapplied science, that pseudoscience in medicine which opposes itself to the clearly ascertainable facts of everyday experience. Hamburger then assailed "that freedom from preconception" which has been such a source of pride. "A real renascence of medical science, on Nazi foundations, must take place." That which is taught by university professors must be completely founded on the tenets of the Nazi program of life and health. "This should be easier for him (the teacher) because national socialism rests on an absolutely sound biologic basis." The chief spokesmen of the various medical disciplines at the universities must be confirmed Nazis; this applies especially to clinicians. Hamburger went on to deprecate the "arrogance of physicians" and to put in a good word for "nature medicine," which, he said, ought not to be too lightly esteemed. "National socialism, unlike any other political philosophy or party program, is in accord with the natural history and biology of man. And because national socialism considers all known physiologic data from nature and from human behavior, it merely represents truths about man. It is accordingly well suited to the direction of the health of our people." Hamburger then turned his fire in succession on Catholicism, liberalism and socialism. He next entered into a discussion of several general problems, in the course of which he said that only a wrongly educated, intellectually biased patient would wish to know the diagnosis—and more along the same line.

Gain in Weight Among Soldiers

The status of body weight is a good measuring rod for the evaluation of the health of youthful persons. Interest therefore attaches to a recent report published by a ranking health officer, Colonel-Surgeon Dr. H. Müller of the sanitary corps of the army. All recruits are weighed during the first half year of their service monthy and later quarterly. The weight is recorded each time in the health statistics of the troops. To obtain an idea of the trends in body weight among young soldiers of the entire reich, statistics on the routine weighings have been compiled by the army health inspector (namely the highest officer in the medical corps in peace time). In the summer of 1938 separate reports were compiled on the recruits who had entered service in November 1937 and soldiers who had already served one year. For both groups the body weight at the time of enlistment was compared with the body weight in the summer of 1938. This represented the first time that such a complete record had been made. The observations were as follows: After the first half year of military training, 70 per cent of the recruits had gained weight, the weight of 10 per cent remained the same and 20 per cent had lost weight. The corresponding figures for men at the end of one year's service were: gained weight 73 per cent, weight stationary 9 per cent and lost weight 18 per cent. It was thus ascertained that in general the tendency to gain weight ends after the first half year. For the entire army the mean increase in weight during the first year amounted to around 3 Kg.

AUSTRALIA

(From Our Regular Correspondent)

March 29, 1939.

Another Plan Replaces National Health Insurance

Following the commonwealth government's decision to abandon its scheme for national health insurance as embodied in the National Insurance Act of 1938, it is intended to substitute a medical service plan. In view of the increased and increasing liabilities incurred on account of defense measures (£500,000 a week in Australia) and the prospective additional cost of liberalizing the national insurance plan as laid down in the act, together with a decreased ability on the part of the government and the people of Australia to meet the cost of maintenance of the scheme, the government has decided to repeal entirely the pension provisions of the act. There remains a system of health insurance and family medical benefit. Details of the scheme have not been determined, but the weekly contribution of 1 shilling sixpence each from employer and employee as provided in the present act is to be reduced approximately by half. It is intended to continue the approved society system and the cash health benefits on substantially the same scale as is now provided. But the whole question of medical benefit is to be discussed first with representatives of medical benefit organizations and of the medical profession and later with the consultative council of approved societies. The announcement that the health service is not to be abandoned makes the matter acute once more for general practitioners in Australia. Now, however, they are well organized to present a united front against governmental aggression and to press their arguments for control of medical services by a board composed mainly of medical men, for the reduction of the income limit for insurance patients, and for the acceptance of certificates and prescriptions from any registered practitioner. It is obvious from a survey of the legislative background in Australia that any hope of finality in the arrangements for "national" insurance is remote.

New Development in Maternity Service

To reduce further maternal mortality figures, the health department of New South Wales has made available better medical facilities for mothers during the puerperal period. Antepartum and postpartum facilities have been set up and are expanding. The scheme now propounded will cover the vital intermediate period. Arrangements include an agreement with the state branch of the British Medical Association whereby the services of accredited obstetric consultants will be made available in any abnormal maternity case, no matter what the financial circumstances of the patient may be, and the establishment of a mobile blood transfusion unit which

will be attached to one of the metropolitan hospitals. Blood donors will be paid at the rate of £5 for every pint of blood taken from them, and the whole of this cost, together with that of bringing in specialists, will be borne by the department of public health. Furthermore, any maternal death occurring will be reviewed within a reasonable time by a special committee, with a view to discovering any loopholes in the scheme and thus to reduce the rate of any similar happening in the future.

Extension of Pharmaceutic Training Suggested

With the advent of New Zealand's social security act of 1938, it becomes imperative that increased laboratory facilities for diagnosis and treatment be made readily available to medical practitioners throughout the country. Rather than bring into existence some new organization, it would be preferable if this were undertaken by an existing body having contact with both the physician and the patient, and for this reason it has been suggested that pharmacists suitably trained in the practice of biochemical analysis would be able to give this service to the state. Such an arrangement insures adequate laboratory services in a form easily available to physician and patient.

The first requisite in any such plan would be to insure an adequate standard among those pharmacists who undertook the work. A diploma of biochemical analysis would certify that the holder was qualified to carry out the work by virtue of his having passed examinations in the subject. Only those pharmacists who held such a diploma would be qualified to carry out this work, and the tests would be performed only on the order of a qualified medical practitioner. As the pharmacist already has the foundations of the required knowledge of general chemistry and of chemical analysis, it appears that he would be the logical person to train for the work.

The scope of the work would include (1) urinary tests and estimations (routine chemical examination, urea concentration test, quantitative analyses for sugar, albumin and chlorides, test of the diastatic activity and fermentative sugar test), (2) blood analyses (dextrose tolerance test, urea estimation and estimation of total nonprotein nitrogen), (3) serum analyses (test for calcium, cholesterol and phosphates, quantitative Van den Bergh test, qualitative Van den Bergh test and protein estimation), (4) fecal analyses (detection of occult blood and estimation of fats) and (5) fractional gastric analyses.

This suggestion has been greeted by various criticisms. While the scheme may look well on paper, practical laboratory experience suggests that there is an element of false security inherent in the proposal. The absence of immediate medical direction for this type of work and the lack of interpretative facilities may lead to error. The range of biochemical work also warrants a turnover, an equipment and a degree of application which are unlikely to be found in even a well equipped pharmacy. The tendency in Australia is to employ a technician of the caliber of a bachelor of science to do this type of work in conjunction with a technician or, sometimes, to combine the two positions when the turnover is limited. But in either case medical direction is given to the work. The suggestion also affects the future of the medical graduate who has specialized in pathology and biochemistry. While the "pathologic unit" working in a well equipped laboratory finds ample scope in towns of a population of about 100,000, a difficulty arises as to what type of unit is suitable for towns and districts of smaller population. Here a better solution of the problem would be the establishment of "pathologic units" at strategically selected centers: the problems of distance between the unit and the scattered practitioners being overcome by modern methods of communication and transport, including the airplane. The pharmaceutic chemist could still be used, but over a much more limited range of technical activities.

BELGIUM

(From Our Regular Correspondent)

Feb. 16, 1939.

Congress of Neurology and Psychiatry

The Congress of Neurology and Psychiatry, organized by neurologic and psychiatric societies of Belgium and the Netherlands, held its sessions at Ghent and at Brussels under the chairmanship of Prof. C. U. Ariëns Kappers. In the first paper the literature of dementia praecox and the encephalitides was reviewed. Dr. Guillaume Vermeylen said that numerous cases of psychoses and notably of dementia praecox have as their basis an encephalitis. These encephalitides often develop insidiously and their nature is as yet poorly defined. They may be the result of a direct influence of the virus or of a state of asthenia which permits a neurotropic virus to become virulent. Moreover, these divergent hypotheses assume a diminution of resistance in the hemato-encephalic barrier. The more highly developed the patient's psychosis, the more diffuse is the cerebral process; and the slower the development of the process, the greater the chances that organized psychoses will be observed to develop.

The second paper dealt with parenchymatous disorders of the cerebellum. Bernard Brouwer and Arie Biemond hold the opinion that the cerebellum is an organ of association for the proprioceptive system and consequently an organ of coordination, above all for the involuntary movements. The activity of the cerebellum renders the movements quicker and easier, since at the cerebellar level no associations of another order come to disturb its functioning, as happens at the level of the hemispheres. The latter may interfere at any time with the physiologic activity of the cerebellum and thus modify the mechanism of motility, but they cannot completely replace the cerebellum in this function.

Occupational Dermatoses

Dr. Henri Halkin has published in the *Liége médical* an article on occupational dermatoses. Certain dermatoses are truly based on the occupation proper, whereas the environment is of minor importance in their manifestation. The principal disorders are due to cement and lime in general, to chromium and the chromates, and to concentrated acids or alkalis, and to globules of oil. Nearly 80 per cent of cutaneous accidents are occupational dermatoses, cutaneous reactions in Brocq's sense of the term. However, in addition to an external cause, there usually is a general disturbed condition of the patient's equilibrium. This concept may be invalidated as soon as more information has been obtained with regard to the phenomena of sensitization.

Ernest Malvoz

Dr. Ernest Malvoz, the recently deceased professor of bacteriology at the University of Liége, was a pioneer in social medicine. For him the highest humanitarian duty was "to combat the three greatest scourges: ignorance, poverty and disease."

Marriages

MARION KEMPER HUMPHRIES JR., Charlottesville, Va., to Miss Zaida Pettit Thomas of Wytheville, April 15.

ARTHUR C. JOHNSON, Omaha, to Mrs. Lotus Hulsebus of Pasadena, Calif., at Las Vegas, Nev., February 17.

CLEMENT P. O'NEILL, Rock Island, Ill., to Miss Baptista Hummer of Davenport, Iowa, April 10.

RUSSELL LEE NORBURN to Mrs. Hope Robertson Colthup, both of Asheville, N. C., April 10.

CHARLES SCOTT MILLER to Miss Ethel Atkinson, both of Philadelphia, recently.

JACOB SALVATORE DIGATE to Miss Edith Alberts, both of Chicago, March 18.

HARRY SHUBIN to Miss Celia Fierman, both of Philadelphia, July 9, 1938.

Deaths

Arthur H. Harrington ⊕ Providence, R. I.; Jefferson Medical College of Philadelphia, 1882; an Affiliate Fellow of the American Medical Association, past president of the Rhode Island Medical Society; member of the American Psychiatric Association and the New England Society of Psychiatry; medical director of the Bridgewater (Mass.) State Hospital, 1894-1898, superintendent of the Danvers (Mass.) State Hospital, 1898-1903, New York Eye and Ear Infirmary, 1903-1907, and State Hospital for Mental Diseases, Howard, 1907-1926; aged 82; died, March 12, of arteriosclerosis and coronary occlusion.

John W. Burns ⊕ Cuero, Texas; Vanderbilt University School of Medicine, Nashville, Tenn., 1891; member of the House of Delegates of the American Medical Association, 1922-1932, 1934-1938, and since 1937 member of the Judicial Council; past president of the State Medical Association of Texas and the De Witt County Medical Society; fellow of the American College of Surgeons; formerly member of the state board of health; medical director of a hospital bearing his name; aged 72; died, May 1.

Charles B. Barker, Guthrie, Okla.; Chicago College of Medicine and Surgery, 1912; member of the Oklahoma State Medical Association and the American Academy of Ophthalmology and Oto-Laryngology; fellow of the American College of Surgeons; aged 54; died, February 12, in St. Anthony Hospital, Oklahoma City, of streptococcic septicemia.

Martin Donelson ⊕ Medical Inspector Commander, U. S. Navy, retired, Danville, Va.; University of Virginia Department of Medicine, Charlottesville, 1906; member of the American Urological Association; entered the navy in 1907 and retired in 1935 for incapacity incident to the service; aged 56; was found dead, February 9, of a bullet wound.

Jules Baechler, West New York, N. J.; Columbia University College of Physicians and Surgeons, New York, 1907; member of the Medical Society of New Jersey; for many years medical examiner for the public schools; aged 61; died, February 11, in the North Hudson Hospital, Weehawken, of cerebral hemorrhage and hypostatic pneumonia.

Lester Jarvis Benson, Lakewood, Ohio; Chicago Homeopathic Medical College, 1903; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1906; honorary member of the staff of the Lakewood City Hospital; aged 78; died, February 12, of acute pulmonary edema and osteoarthritis.

William Chestnut, Winnipeg, Man., Canada; Manitoba Medical College, Winnipeg, 1898; professor emeritus of medicine at his alma mater; for many years on the honorary attending staff and formerly medical superintendent of the Winnipeg General Hospital; aged 71; died, February 7, of coronary occlusion.

Cornelia Bernhardine Johanna Schorer ⊕ Foxboro, Mass.; Universität Zürich Medizinische Fakultät, Switzerland, 1897; an Affiliate Fellow of the American Medical Association; member of the American Psychiatric Association; formerly on the staff of the Foxboro State Hospital; died, January 9, in Berlin, Germany.

Reynolds May, Whitewright, Texas; University of Louisville (Ky.) Medical Department, 1878; member of the State Medical Association of Texas; past president of the Grayson County Medical Society; formerly member of the board of education; aged 87; died, January 2, in a hospital at Sherman of heart disease.

Reuben C. Burrow, Cunningham, Ky.; Vanderbilt University School of Medicine, Nashville, Tenn., 1891; Eclectic Medical Institute, Cincinnati, 1894; member of the Kentucky State Medical Association; past president and secretary of the Carlisle County Medical Society; aged 70; died, February 2, of pneumonia.

Walter Barwick Bruce, Helena, Ark.; University of the South Medical Department, Sewanee, Tenn., 1901; member of the Arkansas Medical Society; county health officer; for many years member of the board of trustees of the University of the South, Sewanee, Tenn.; aged 65; died, February 22, of cerebral hemorrhage.

Amos H. Caffee ⊕ Terre Haute, Ind.; Central College of Physicians and Surgeons, Indianapolis, 1900; past president of the Vigo County Medical Society; secretary of the city board of health; served during the World War; on the staff of the Union Hospital; aged 73; died, February 1, of coronary thrombosis.

Frank Joseph Alessi, Niagara Falls, N. Y.; University of Buffalo School of Medicine, 1929; on the staffs of the Memorial Hospital and St. Mary's Hospital, Niagara Falls, and the Millard Fillmore Hospital, Buffalo; aged 34; died, January 10, in Saranac Lake of pulmonary tuberculosis.

Justin M. De Ford, Rossburg, Ohio; Cincinnati College of Medicine and Surgery, 1892; member of the Ohio State Medical Association; for many years member of the local board of education and at one time member of the county board of education; aged 82; died, March 4, of pneumonia.

William Heilman Hossler, Staten Island, N. Y.; Medical College of Ohio, Cincinnati, 1907; formerly acting assistant surgeon in the United States Public Health Service, Immigration Department; aged 59; died, January 13, in the United States Marine Hospital of diabetes mellitus.

Edward Homer Egbert, St. Simon Island, Ga.; Baltimore Medical College, 1905; member of the Medical Association of Georgia; served during the World War; formerly surgeon to the Huggins Hospital, Wolfeboro, N. H.; aged 57; died, February 27, of coronary thrombosis.

John H. Fletcher, Wichita Falls, Texas; St. Louis College of Physicians and Surgeons, 1905; veteran of the Spanish-American and World wars; city health officer and formerly county health officer; aged 60; died, January 29, in a local hospital of cirrhosis of the liver.

Robert Jasper Alexander ⊕ Waco, Texas; Memphis (Tenn.) Hospital Medical College, 1891; fellow of the American College of Surgeons; on the staffs of the Providence and Central Texas Baptist sanitariums; aged 72; died, February 20, of acute hypostatic pneumonia.

Philip James Reichmann, Oakwood, Mo.; St. Louis College of Physicians and Surgeons, 1911; member of the Missouri State Medical Association; aged 61; died, January 11, in St. Elizabeth's Hospital, Hannibal, of diabetes mellitus and gangrene of the left foot.

William Thomas Asher, Atlanta, Ga.; Atlanta Medical College, 1893; member of the Medical Association of Georgia; on the staffs of St. Joseph Infirmary and the Grady Hospital; aged 67; died, February 7, of cerebral thrombosis and cerebrovascular degeneration.

Alfred Mitchell Merriman ⊕ Bristol, R. I.; Medical School of Maine, Portland, 1895; at one time medical inspector of the public schools; formerly on the staff of the Rhode Island Soldiers' Home; aged 70; died, January 9, in the Jane Brown Hospital, Providence.

John Norris Doyle, Wausau, Wis.; Marquette University School of Medicine, Milwaukee, 1915; member of the State Medical Society of Wisconsin; served during the World War; aged 53; died, February 4, in a hospital at Temple, Texas, of pneumonia.

Charles Daligny, Troy, N. C.; Université de Paris Faculté de Médecine, France, 1878; member of the Medical Society of the State of North Carolina; served in the Franco-Prussian War; aged 85; died, January 8, of carcinoma of the prostate and uremia.

Howard Newell Baker, Pierson, Iowa; Hahnemann Medical College and Hospital, Chicago, 1905; member of the Iowa State Medical Society; formerly a member of the board of education; aged 57; died, February 8, of cerebral hemorrhage.

Charles Joseph King, Langdon, N. D.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1904; Dearborn Medical College, Chicago, 1906; aged 59; died, January 31, of coronary occlusion.

Chester Roy Brigham, Polo, Ill.; Keokuk (Iowa) Medical College, College of Physicians and Surgeons, 1905; member of the Illinois State Medical Society; served during the World War; aged 66; died, February 27, of coronary thrombosis.

Clarence Hathorne Staples, Malden, Mass.; Harvard University Medical School, Boston, 1904; member of the Massachusetts Medical Society; on the staff of the Malden Hospital; aged 60; died, January 17, of cerebral hemorrhage.

Marcus James Greenlee, Sewickley, Pa.; Howard University College of Medicine, Washington, D. C., 1937; aged 28; died, January 24, in the Suburban General Hospital, Bellevue, of skull fracture received in an automobile accident.

Lincoln Riegel Light, Lebanon, Pa.; Jefferson Medical College of Philadelphia, 1892; member of the Medical Society of the State of Pennsylvania; aged 71; died, January 12, of cerebral hemorrhage, arteriosclerosis and hypertension.

Archibald D. Campbell, Richland Center, Wis.; Louisville (Ky.) Medical College, 1896; aged 73; on the staff of the Richland Hospital, where he died, February 18, of bronchopneumonia, arteriosclerosis and chronic myocarditis.

John James Lindsay, Spartanburg, S. C.; University of Maryland School of Medicine, Baltimore, 1887; member of the South Carolina Medical Association; aged 73; died, January 5, at the Spartanburg General Hospital.

Edward Joseph Howland ✠ Colchester, Conn.; University of Vermont College of Medicine, Burlington, 1911; served during the World War; for many years health officer; aged 58; died, January 25, of coronary thrombosis.

Lee H. Duguid, Michigan Center, Mich.; Kansas Medical College, Medical Department of Washburn College, Topeka, 1898; served during the World War; aged 66; died, January 8, in the United States Marine Hospital, Detroit.

William Frederick Vilter, Cincinnati; Medical College of Ohio, Cincinnati, 1901; member of the Ohio State Medical Association; on the staff of the Deaconess Hospital; aged 62; died, January 21, of lymphatic leukemia.

Frank Edward Tompkins, Brooklyn; Columbia University College of Physicians and Surgeons, New York, 1899; aged 62; died, January 1, in the Boro Park General Hospital of chronic nephritis and uremia.

Thomas Francis Shinnick, Beloit, Wis.; Rush Medical College, Chicago, 1899; member of the State Medical Society of Wisconsin; served during the World War; aged 65; died, January 29, of heart disease.

John Stephen Thompson, Winterport, Maine; Georgetown University School of Medicine, Washington, 1895; aged 75; died, January 8, in Antigonish, N. S., of anemia and acute dilatation of the heart.

Charles Lewis Hawkins ✠ Taft, Calif.; Jefferson Medical College of Philadelphia, 1900; member of the American Urological Association; aged 68; died, January 14, of abscess about the ampulla of Vater.

Ada Scott Conner Morton, Fall Brook, Calif.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1907; aged 59; died, January 11, of angina pectoris.

William Lincoln Shawk, Rockaway Beach, Calif.; Western Reserve University Medical Department, Cleveland, 1883; aged 78; died, January 7, of arteriosclerosis and cerebral hemorrhage.

Winfield Howard Ames, Milford, N. H.; Syracuse University College of Medicine, 1893; aged 70; died, January 25, of abscess of the axilla following injury by crutch, and hemophilia.

Ross David Long, Oklahoma City; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1899; aged 69; died in January of cerebral arteriosclerosis.

Joe Johnson Taylor, Riverside, Calif.; American Medical College, St. Louis, 1893; St. Louis College of Physicians and Surgeons, 1908; aged 72; died, January 6, of chronic myocarditis.

Jefferson Davis Duke, Heflin, Ala.; Southern Medical College, Atlanta, Ga., 1884; member of the Medical Association of the State of Alabama; aged 77; died, February 12, of hemiplegia.

William Patrick Brogan, Tyler, Texas (licensed in Texas, under the Act of 1907); member of the State Medical Association of Texas; aged 67; died, January 6, of arthritis deformans.

Charles Leona Badger, Indianapolis; Indiana University School of Medicine, Indianapolis, 1911; aged 60; died, February 5, in St. Vincent's Hospital of an overdose of a narcotic.

Kenneth Paul Hunter, Wapello, Iowa; State University of Iowa College of Medicine, Iowa City, 1927; served during the World War; aged 39; died, January 15, of heart disease.

Frank Milton Hamblin, Bristol, Conn.; New York Homeopathic Medical College and Hospital, 1899; aged 65; died, January 30, in a hospital at Middletown of arteriosclerosis.

James Johnson Ellis, Knoxville, Tenn.; Tennessee Medical College, Knoxville, 1899; member of the Tennessee State Medical Association; formerly state senator; aged 80; died, January 5.

John A. P. Shields, Hartford, Tenn.; Chattanooga Medical College, 1902; member of the Tennessee State Medical Association; formerly state senator; aged 69; died, January 17.

David C. Trach ✠ Kresgeville, Pa.; College of Physicians and Surgeons, Baltimore, 1891; aged 71; died, January 8, in the University of Pennsylvania Hospital, Philadelphia.

Nelson Du Val Brecht, Washington, D. C.; George Washington University School of Medicine, Washington, 1906; aged 54; died, February 15, of a self-inflicted bullet wound.

Joseph McClanahan Cornett, Temple, Texas; Baylor University College of Medicine, Dallas, 1934; intern at the Scott and White Hospital; aged 24; died in January.

Edward Sax Drucks, Oakland, Calif.; College of Physicians and Surgeons of San Francisco, 1907; member of the California Medical Association; aged 55; died, January 9.

James Krauss, Boston; Boston University School of Medicine, 1889; fellow of the American College of Surgeons; aged 72; died, January 29, of bronchopneumonia.

Joseph Price Dancy, Detroit; Howard University College of Medicine, Washington, D. C., 1920; aged 44; died, February 13, in the Parkside Hospital of meningitis.

Frank Joseph Doyle, Baltimore; Georgetown University School of Medicine, Washington, D. C., 1901; aged 60; died, February 5, of valvular heart disease.

Ellis Reynolds Shipp, Salt Lake City; Woman's Medical College of Pennsylvania, Philadelphia, 1878; aged 92; died, January 31, of carcinoma of the neck.

William S. Manners, San Leandro, Calif.; Northwestern Ohio Medical College, Toledo, Ohio, 1888; aged 82; died, January 28, of chronic myocarditis.

James H. West, McCrory, Ark. (licensed in Arkansas in 1905); aged 59; died, January 29, in the Baptist Hospital, Memphis, Tenn., of heart disease.

Calvin S. De Voll, Fort Worth, Texas; Metropolitan Medical College, Chicago, 1900; aged 86; died, February 19, of uremia and cardiorenal disease.

Osborne Elliott Carr, Owen Sound, Ont., Canada; University of Toronto Faculty of Medicine, 1913; aged 52; died, February 23, of heart disease.

Julian Philip M. Johnson, Yuba City, Calif.; State University of Iowa College of Medicine, Iowa City, 1910; aged 65; died, January 25, in India.

Jacob Koronefsky ✠ New York; Columbia University College of Physicians and Surgeons, New York, 1900; aged 69; died, January 16.

John Clemons Lancaster, Houston, Texas (licensed in Texas under the Act of 1907); aged 87; died, January 31, of bronchopneumonia.

Alice Maud Gray, Boston; Tufts College Medical School, Boston, 1900; member of the Massachusetts Medical Society; died, January 17.

Vincent Millasich, Fresno, Calif.; California Medical College, San Francisco, 1906; aged 81; died, January 28, of cerebral hemorrhage.

Ulysses S. Ammerman, Reynolds, Neb.; Keokuk (Iowa) Medical College, 1898; aged 74; died, January 19, of coronary thrombosis.

William Noble Nanney, Whittier, Calif.; Medical College of Evansville, Ind., 1878; aged 85; died, January 12, of chronic myocarditis.

George Henry Baker, Long Branch, N. J.; Albany (N. Y.) Medical College, 1886; aged 84; died, February 8, of bronchopneumonia.

Stuart Eccles Beech, Salmon Arm, B. C., Canada; Manitoba Medical College, Winnipeg, 1911; aged 54; died, January 29.

William O. Curlee, Lubbock, Texas (licensed in Texas, under the Act of 1907); aged 62; died, February 7, of coronary disease.

Stephen Sinclair Slauenwhite, Windsor, N. S., Canada; Halifax (N. S.) Medical College, 1896; aged 72; died, January 12.

Arthur E. Fraleigh, St. Mary's, Ont., Canada; Western University Faculty of Medicine, London, 1892; died, January 17.

Morton Lake Hooper, Goldsboro, N. C.; Baltimore Medical College, 1887; aged 81; died, January 22, of chronic myocarditis.

John W. J. Newman, Buchanan, Ga.; Atlanta Medical College, 1893; aged 73; died in January of heart disease.

Charles H. A. Meyer, Baltimore; Baltimore Medical College, 1888; aged 78; died, January 1, of heart disease.

Marjory Ward, Renfrew, Ont., Canada; Trinity Medical College, Toronto, 1894; aged 68; died, January 25.

Robert G. Turner, Nacogdoches, Texas (licensed in Texas, under the Act of 1907); aged 62; died, January 2.

Gardner Spring Chapin, Los Angeles; Rush Medical College, Chicago, 1903; aged 59; died, January 1.

Correspondence

A PROGRAM FOR THE REFUGEE PHYSICIAN

To the Editor:—The purpose of this statement is to inform the medical profession at large of the facts concerning refugee physicians in this country and to offer a just and reasonable program to resolve the existing problem to the mutual advantage of the communities in which they are seeking shelter and of the émigrés themselves.

Until recently current misconceptions about the facts and a lack of understanding of the nature of the situation have added needless difficulties and complications. The committee hopes that this statement will serve primarily to clarify the facts in order to provide a better spirit of understanding in terms of mutual responsibility.

THE FACTS

During the period from July 1, 1934, to Dec. 1, 1938, corresponding to the time of the National Socialist régime in Germany, 1,528 physicians have migrated to the United States from Greater Germany.

The official figures covering this period are the following:

July 1, 1934, to July 1, 1935	104
July 1, 1935, to July 1, 1936	253
July 1, 1936, to July 1, 1937	286
July 1, 1937, to July 1, 1938	365
July 1, to Dec. 1, 1938	520
	1,528

Of this number, approximately 25 per cent are Catholic or Protestant and 75 per cent are of Jewish or partly Jewish origin.

There are at present 170,000 physicians in the United States, and our own medical schools graduate 5,500 new physicians each year. Despite this, the number of physicians in relation to the general population has been steadily decreasing, from 1 to 662 in 1886 to 1 to 784 in 1934.

THE PROBLEM IS SELF LIMITED

On the basis of the situation abroad, there will be a substantial reduction in the number of refugee physicians entering this country in future years, since:

1. Those who now apply for admission to this country must wait three or four years for their quota numbers to become available.

2. An indication of the limited nature of the problem is furnished by official Berlin statistics, published in the Sept. 17, 1938, issue of THE JOURNAL, revealing that there were fewer than 2,500 Jewish doctors left in Germany proper on July 1, 1938. Of these some have since emigrated to all parts of the globe. Approximately 700 have permits to practice among the Jews left in Germany. It is estimated that there are hardly more than 1,000 potential medical emigrants in Germany itself. In this connection, an item in the New York *Times* of April 26, 1939, dealing with Jewish emigration from Germany, is of particular interest: "Significantly enough, the only Jews whose emigration is being discouraged are physicians, especially surgeons, who might prove useful in case of war."

The quotas for admission to the United States from Poland, Hungary, Rumania and Czechoslovakia are so small that in any one year not more than an aggregate of 200 to 300 physicians could possibly enter from these countries.

3. Many of the Jewish physicians remaining in Germany are advanced in years. Many are close to the age of retirement.

4. Students of the type who later might have to emigrate can no longer enter the medical schools in Greater Germany, Czechoslovakia, and so on. In the eastern European countries, restriction of entry for such students in medical schools has been in practice for years, and we need anticipate no significant immigration of physicians into the United States from these countries.

The peak load has clearly been passed. The concentration of the refugees on the Eastern Seaboard has created an exaggerated impression of their actual numbers, either present or future. Therefore the committee feels that the question has now become one of the proper selection and distribution of a limited number of physicians and that an adequate and practical solution can be found based on a carefully studied and administered plan.

THE STRUCTURE

The Committee for the Resettlement of Foreign Physicians has been organized under the egis of the nonsectarian National Coordinating Committee for Aid to Refugees. Since New York City is the port of entry for the large majority of immigrants, the central office is located at 165 West Forty-Sixth Street. However, there is a national committee composed of representatives of members of the local committees, such as the Boston Committee on Medical Emigrés (Dr. David Edsall, chairman), the Baltimore committee (Dr. John M. T. Finney, chairman) and the Chicago committee (Dr. Dallas B. Phemister, chairman), as well as of the New Haven and New Jersey committees, which are being formed. The New York committee works in close cooperation with these committees, but since the initial task of selection devolves on New York, the following organization has been set up:

Members of the General Advisory Council include Dr. George Baehr, Dr. Clarence G. Bandler, Dr. Ernst P. Boas, Dr. Louis Casamajor, Dr. Alfred E. Cohn, Dr. Lewis A. Conner, Dr. Haven Emerson, Dr. N. Chandler Foot, Dr. Frank Fremont-Smith, Dr. Irving Graef, Dr. Foster Kennedy, Dr. John D. Kernan, Dr. Lawrence S. Kubie, Dr. Emanuel Libman, Dr. Currier McEwen, Dr. Walter W. Palmer, Dr. Henry Aslop Riley, Dr. Peyton Rous, Dr. Charles Hendee Smith, Dr. J. Bentley Squier, Dr. Mills Sturtevant, Dr. Homer F. Swift, Dr. Howard C. Taylor Jr. and Dr. I. Ogden Woodruff.

There is a central office, where each refugee physician is registered for the detailed examination of his credentials. (Information about any foreign physician may be obtained by writing to Mr. Charles H. Jordan, Secretary.)

There are advisory boards in each of twenty medical specialties whose function it is to evaluate and examine the personal and scientific qualifications of every refugee. These boards make confidential reports on the qualifications of each individual physician, which are filed in the central office.

Those who are passed by the advisory board are then given material aid by the National Coordinating Committee while studying for state board examinations.

A curriculum committee arranges lectures in various departments of medicine. Emphasis is placed on the broad cultural requirements of American medicine as well as on the factual information required for the licensing examinations. A program for clinical retraining is being prepared to fit certain specialists for general practice.

The committee seeks to secure voluntary assistantships, supernumerary internships and temporary clinic appointments for eligible physicians to assist them in their studies.

After passing their examinations, physicians are resettled in communities where there is a known shortage of medical care. This is done only after consultation with the local authorities and with local representatives of the medical profession.

THE SOLUTION

Community needs must be considered as well as the needs of the physician. Resettlement of qualified physicians in areas most in need of medical service, and therefore best able to absorb the newcomers, is the solution proposed by the Committee for the Resettlement of Foreign Physicians. To this end the committee hopes that a change can be effected in the regulations in a majority of states, which now either bar foreign physicians from practice or make it unduly difficult for them to obtain a license until they become citizens (a period of five years). These local regulations have been largely responsible for the concentration of these physicians on the Atlantic seaboard.

Unquestionably certain foreign physicians cannot and should not be permitted to practice here until they are thoroughly qualified. A few will never be able to meet American requirements. These, obviously, must be retrained outside the medical field.

The committee proposes, through an examining board of qualified physicians, to pass upon the personal or scientific qualifications of the incoming physicians. It will give no support to those who are adjudged to be unfit to practice in this country.

In this connection it is important to quote here from a statement of the Boston Committee on Medical Emigrés, as follows:

After some ten months of experience, this Committee has learned:

1. That a substantial proportion of the medical refugees here and abroad are among the most talented members of the profession, in research and in clinical fields.

2. That existing prejudices against them derive most often from lack of contact and from fear that as aliens they are somehow undesirable. When they are given an opportunity to present themselves in person and have their professional achievements properly scrutinized, preconceived objections to them frequently soften or entirely disappear.

3. It appears likely that, if a thoroughgoing search of this country's unfilled medical needs were made, all of these men who have arrived or may arrive here and whom we consider competent would be absorbed without difficulty and to the distinct advantage of institutions and communities which otherwise go without the services they can provide.

It is the hope of the Committee for the Resettlement of Foreign Physicians that all members of the medical profession will cooperate in its work, to the end that the refugee physicians, who have no desire to compete with American physicians in congested areas, may be given an opportunity to exercise their profession, their skill and their training in areas where their services are most needed.

DAVID L. EDSALL, M.D., Boston.
Honorary Chairman, National Committee for the Resettlement of Foreign Physicians.

EFFECT OF SUNLIGHT ON BONE AS REPORTED BY HERODOTUS IN 450 B. C.

To the Editor: — In book III of Herodotus's history, in which he describes some of his travels in Egypt and Libya, an account is given of the Persian invasion of Egypt under Cambyses. Near the mouth of the so-called Pelusian Nile, the forces of Cambyses met the Egyptians under King Psammenitus in 525 B. C. Herodotus viewed the battle field about seventy-five years later. His attention was called to the striking difference in thickness and strength of the Persian and Egyptian skulls which were still lying on the surface of the ground. His explanation of this difference is interesting, being so far as I know the earliest recorded definite proof offered of the effect of sunshine in thickening and strengthening bone tissue. A translation of chapter 12 of book III, in as nearly the simple style of the original Ionic Greek as I can make it, runs as follows:

"I saw a great marvel of which I had heard from the natives of the country. The bones of those who fell on either side in this battle lay scattered separately. The bones of the Persians lay in one part of the field and the bones of the Egyptians in another, as the two armies had separately stood. The skulls of the Persians were so fragile that a mere pebble thrown at them would penetrate them. But those of the Egyptians were so strong that you could hardly break them with a stone. The cause of this, so the people said, and I readily agreed, is that from childhood the Egyptians shave their heads, and the bone is thickened by exposure to the sun. For the same reason they do not become bald. Of all races of men bald heads are rarest among the Egyptians. Such then is the reason for their strong skulls. And the reason why the Persians have weak skulls is that they cover their heads all their lives with felt hoods, called tiaras by the Persians. Such are the facts of the case. I noticed the same condition at Papremis in the skulls of the Persians slain by Inaros the Libyan."

E. V. WILCOX, Chevy Chase, Md.

"DIAPHRAGMATIC FLUTTER WITH SYMPTOMS OF ANGINA PECTORIS"

To the Editor:—It may be of interest to your readers to add a note as to the peregrination of the medical curiosity reported in THE JOURNAL by Dr. W. B. Porter March 21, 1936, page 992, and by Drs. Whitehead, Burnett and Lagen April 1, 1939, page 1236, as a case of diaphragmatic flutter with symptoms of angina pectoris.

The patient was brought to the Massachusetts General Hospital Oct. 27, 1937, by police ambulance with the identical clinical picture as described by the previous writers. He lay doubled up on his right side, moaning in pain and in no respiratory distress, with a pulse of 80 and a blood pressure 130/80. He was not perspiring and his color was good. A rapid flutter could be felt and ausculted over the precordium and abdomen, and could be seen as a ripple over the latter. Fluoroscopy showed a normally beating heart and a diaphragm that oscillated with about a 1 cm. excursion at a very rapid rate. An electrocardiogram showed a regular oscillation of the base line of 1 mm. amplitude at a rate close to 300 per minute. His temperature was normal.

He gave his name as Frank Crowley and his age as 65. On entry he presented us with a reprint of Dr. Porter's article, so I presume that is why he listed his occupation as a deep-sea diver, as he did in Richmond, Va. Numerous puncture marks were noticed over both antecubital spaces and his speedy release from all pain on entry after one-fourth grain (0.016 Gm.) of morphine and his subsequent loud demands for more gave some support to the belief that he was addicted to drugs. He proved a very irascible patient and demanded release at daylight on the morning after entry, which was speedily granted after threats to nurses. A psychiatrist thought that he was "queer" but probably not psychotic.

The next day he was picked up from the side of the road by a passing mortorist and was last heard from at the Framingham Hospital.

He was an excellent raconteur and his stories of inspecting the battleship *Maine* and the *Lusitania*, and of crossing the floor of the Red Sea in search of chariots were greatly appreciated.

EDWARD HAMLIN JR., M.D., Boston.

"SYPHILIS IN NORTH CAROLINA"

To the Editor:—In THE JOURNAL April 1, page 1260, appears a short comment entitled "Syphilis in North Carolina." In this article you raise a doubt as to the accuracy of the statement to which you refer and intimate that this statement was made for the purpose of securing federal funds for the control of syphilis in this state.

It is felt in this department that you have placed your own interpretation on these figures and evidently have arrived at an erroneous conclusion. You have mistaken the prevalence of syphilis for the incidence of syphilis. The 48,000 "new cases of syphilis" reported each year are not cases of "new" syphilis in the sense that the disease was acquired within the year in which it was reported. These "new" cases mean simply that they have never been reported before to this department. Many of them are cases of latent and late syphilis, but from a reporting standpoint they are new in that they have never previously been brought to the attention of this department by case report. From your comments you evidently interpreted the 48,000 cases to be those of newly acquired syphilis, acquired within the year in which they were reported. By actual count since Jan. 1, 1936, to Dec. 31, 1938, we have had reported to this department 164,254 cases of syphilis, and you can readily see that this is in excess of 4,000 "new" cases per month.

CARL V. REYNOLDS, M.D., Raleigh, N. C.
Secretary, North Carolina State Board of Health, and State Health Officer.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

POSSIBLE ACUTE CARBON MONOXIDE POISONING

To the Editor:—I was called to attend a patient in coma. The man, aged 24, was apparently healthy with no history of previous serious illness. He states that prior to becoming unconscious he operated the motor of a large truck in a garage with the doors open. In about a half hour he went to the second floor of the same building and looked at the clock; he expected a telephone call and definitely recalled the time. He then attempted to open a window but fell to the floor and recalled nothing until an hour and twenty minutes later, when I saw him. His face appeared flushed with no cyanosis except slight cyanosis of the lobules of the ears. He seemed to recognize me but was unable to speak. There was a definite flaccid paralysis of the left arm and leg. The deep reflexes of both legs and arms were exaggerated. There was no ankle clonus nor was the Babinski reflex present. The tongue deviated slightly to the left, and the left corner of the mouth was slightly raised. The right pupil was somewhat larger than the left, and this condition persists. Both pupils reacted well to light. The heart rate was 84 and there was arrhythmia. On admission to the hospital the temperature was 100 F. Despite the fact that I noted automobile fumes in the room and had all the windows opened, I thought that the patient had suffered a cerebral embolus. The blood pressure was 120 systolic, 82 diastolic. Within four hours the patient apparently completely recovered. He spoke clearly and the foregoing history was elicited. He could use the left arm and leg well. The pulse rose to 120 and the temperature fell to 98 F. and then to 97 F. in twelve hours. On the third day the pulse fell to 60 and then on the following day to 52. The blood pressure rose to 136/88. The pulse rate one week later was 72. The patient complained of headache shortly after recovering consciousness, but this persisted for only three days. Urinalysis revealed dark green urine, alkaline in reaction, with a specific gravity of 1.032; it was negative for sugar and albumin but the specimen contained 1 plus pus cells. The Kahn test gave negative results; examination of the blood showed red blood cells 4,990,000, hemoglobin 100 per cent, leukocytes 13,100, polymorphonuclear neutrophils 94 per cent, basophils 1 per cent, small lymphocytes, 4 per cent, large lymphocytes 1 per cent. Repeated urinalyses were negative for sugar. I have thought that this patient was overcome by carbon monoxide and have so stated. Are the foregoing symptoms and signs, together with the history, in harmony with such a diagnosis? The patient seems perfectly well but is somewhat anxious about having a recurrence of such an attack if not due to carbon monoxide poisoning.

M.D., Pa.

ANSWER.—On the basis of the data submitted, which contain no real evidence of exposure to carbon monoxide other than the operation of a motor in a large garage with open doors, one cannot make a definite diagnosis of carbon monoxide poisoning. There are no statements as to the effects on other men in the garage at the same time and no data as to possible concentrations of carbon monoxide in the air. Under the circumstances of this accident, the conditions could readily be duplicated and such information obtained. If it could be shown that the air contained from 600 to 800 parts of carbon monoxide per million parts (or from 0.06 to 0.08 per cent), it would probably be proper to assume quite definitely that the illness was due to carbon monoxide. At the present time, far too many conditions are attributed to carbon monoxide without proper reference to the circumstances of exposure, and these should and generally can be proved.

If the symptoms are considered alone and if it is assumed that there is definite evidence that carbon monoxide was in the atmosphere, it is quite fair to believe that this is a case of carbon monoxide asphyxia. Almost any variety of acute neurologic disturbance can occur in carbon monoxide poisoning, and it is frequent that unconsciousness or some serious neurologic symptom is experienced when the patient attempts to move about actively, even though he may move to an atmosphere which is free of carbon monoxide. A man poisoned by carbon monoxide has been brought to the limit of his power to supply his tissues with oxygen from the blood. "He can stand no extra strain of any sort and, even when he regains consciousness, as may be the case when the blood carbon monoxide reaches 50 per cent of saturation, he still has so slight a margin of oxygen tension as to be quite unequal to activity of any sort without suffering instantly from oxygen lack. As Stadie and Martin (1925-1926) remark: 'Despite the presence in the blood of two or three times the amount of oxygen necessary for normal tissue function, profound anoxemia results in CO poisoning. This is due to an alteration of the shape and position of the oxygen dissociation curve causing a marked lowering of the partial pressure at which oxygen is available for tissue metabolism'" (Drinker, C. K.: Carbon Monoxide Asphyxia, New York, Oxford University Press, 1938. Stadie, W. C., and Martin, K. A.: The Elimination of Carbon Monoxide from the Blood: A Theoretical and Experimental Study, *J. Clin. Investigation* **2**:77 [Oct.] 1925). It is the usual rule that those who have had acute experiences due to carbon monoxide such as this recover completely and there is, of course, no reason why the condition should leave any threat behind in terms of possible further trouble.

URTICARIA AND MENSTRUATION

To the Editor:—Please give me some information as to the cause, treatment and prognosis of menstrual urticaria. The patient is about 25 years of age and ten months ago she had a cesarean section. Her menstruations are sometimes from four to seven weeks apart, but the history of menstrual urticaria is of several years' duration and the condition lasts from three to five days.

H. C. WIGER, M.D., Rice Lake, Wis.

ANSWER.—It is presumed that by "menstrual urticaria" the inquirer has reference to urticarial attacks of a generalized nature (not localized to lesions on the skin in the proximity of the genitalia) occurring particularly in the premenstrual or menstrual periods. A history of urticaria or other allergic manifestations appearing solely or more frequently at that time is a common part of the patient's story. A number of medical articles dealing with that subject have been published. Almost without exception they are all clinical and based on consulting room observation. Intensive studies of a laboratory and experimental nature which might throw light on the association of these two factors, allergy and endocrine, are still lacking. It may be mentioned that there are other associations of a possible relationship between allergy and the endocrines. Some of these are exemplified by the tendency of allergic symptoms to be improved at puberty in the boy and to be aggravated in the same period in the girl, by the likelihood for allergic symptoms to diminish or vanish altogether during pregnancy and by the frequent change in the course of clinical allergy at the menopause.

Until the indicated experimental and laboratory work in the future provides a basis for a more specific and definite correlation of allergic symptoms and the menstrual cycle, this relationship will be regarded as a general one. According to the latter conception allergic symptoms are more apt to occur around the menstrual period just as they are more apt to occur following other emotional upsets, infections, exertion and thermal, mechanical and chemical stimuli. They are usually the precipitating factors rather than the specific causes in a patient who has an ever present allergy but is capable of being in allergic balance most of the time. In the case cited and in similar types there are three main methods by which one may achieve results: One is, of course, by a search for the basic underlying allergy. Another is by the use of rest, sedation and such other measures as may counteract the excitability of the nervous system. Finally, results sometimes may be achieved by endocrine therapy. The prognosis is fair.

SILVER PREPARATIONS FOR SINUSITIS

To the Editor:—Please discuss the treatment of sinusitis by the insertion of colloid silver tampons.

M.D., Massachusetts.

ANSWER.—The use of colloidal silver tampons in the treatment of chronic sinusitis is practiced extensively by otorhinolaryngologists in this country. In a recent questionnaire it was shown that approximately 90 per cent of specialists used some silver salt in treating diseases of the ear, nose and throat. Although practically every silver preparation has been used, the favorite one is mild protein silver (argyrol) usually in 10 per cent strength.

Colloidal silver preparations are said to be bactericidal, sedative to inflamed mucous membranes and stimulating to reparative powers. These are debatable points. It is true, however, that the colloidal silver preparations are mildly astringent and it is partly due to this property that they give symptomatic relief. Moreover, because of their colloidal state and their astringent property they do incite a profuse watery secretion from the mucous membranes which depletes the nasal mucosa, thus supposedly helping rid the nose and sinuses of bacteria and pus.

The more ardent proponents of the silver tampon treatment have formulated a routine of tampon insertion, designating types of cotton, applicators and position of the tampon. The latter, the "sphenoid" and "infundibulum" direction, is said to bring

the medication into contact with most of the sinus ostiums and is considered by some to be absolutely indispensable to the full success of the procedure. The tampons are left in place from thirty minutes to an hour and after removal the nose is flushed with some cool irrigating fluid.

Despite the popularity of this form of treatment there are many capable rhinologists who feel that it has no peculiar advantages over many other conservative methods of treating chronic sinusitis.

The bacteriostatic and bactericidal power of mild protein silver as investigated by Hilding, Cannon and Walsh, is so slight as to be of little clinical importance. The astringent effects can be achieved by many other drugs without too elaborate a technic of medication. Though mild protein silver has not been shown to be harmful to ciliary activity, the possibility of argyria, local or general, even though infrequent, must be kept in mind when one is using silver salts extensively.

POSSIBLE HEAT STROKE WITH APHASIA

To the Editor:—The following is the summary of a case of mixed aphasia which suddenly occurred coincidentally with what was diagnosed as "heat stroke." A white man aged 50, large, well developed and muscular, was working digging a basement for a school house. The day was hot, there was no air circulating in the pit and the sun was beaming overhead. At 1 p. m. the patient suddenly got faint and collapsed unconscious. His fellow workmen first thought he had fainted, but when he failed to revive he was taken to the hospital, where he had a subnormal temperature and excessive sweating, and where he remained unconscious for four days. On regaining consciousness he was found to have a motor aphasia, apraxia, paragraphia and partial auditory aphasia but no alexia. Right hemiparesis and right hemiapraxia were present. The tendon jerks were hyperactive on the right side. The spinal fluid was normal, the gold curve 00000000, the Wassermann reaction of both the spinal fluid and the blood was negative. The pressure was 150 mm. initially and also after 10 cc. had been removed. The cranial nerves were entirely normal, there being no evidence of arteriosclerosis of the retinal vessels. The blood pressure was 140 systolic, 90 diastolic. The peripheral arteries were soft. The patient shows no clinical evidence of cardiac, renal or other organic disease. The patient is suing for compensation insurance, none of which has ever been paid since the onset of his illness. Now the question arises Can heat stroke cause aphasic symptoms such as the aforementioned and can you cite such cases on record? I might add that the signs and symptoms have remained unchanged during the past six months since the onset of the illness.

W. L. SHARP, M.D., Anderson, Ind.

ANSWER.—It is improbable that the patient is suffering from heat stroke per se unless it can be shown that he was entirely well without cardiac, renal or any other organic disease prior to the onset of his coma and that following the exposure he developed a deep coma with a temperature of from 104 to 108 F. as well as an increased cell count in the spinal fluid. If this can be done it will be possible to establish a causal relationship between the heat of the sun and the present symptoms. If he had these symptoms (heat stroke), this is one of the rare cases in which recovery occurs. Most all patients with heat stroke or apoplexy die because of the high temperatures and dehydration despite all therapy. It seems peculiar that the man showed only those signs of a large softening or hemorrhage (embolus, hemorrhage or multiple thrombi) on the left side of the brain. In heat stroke there is a widespread ganglion cell damage which produces not only hemiplegia, paraplegia, ataxia and aphasia but also convulsions and psychosis. The article by Shattuck and Hilferty (*Am. J. Trop. Med.* **12**:223 [May] 1932) is recommended for perusal.

NARCOTIC ADDICTION AND HEREDITY

To the Editor:—I should like information concerning the effect of the use of morphine by the father, for a number of years previous to and at the time of conception, on the offspring. I also wish the same information where the father discontinued the use of morphine a few months prior to conception. I would also appreciate general information concerning morphinism and reproduction, especially relating to its effect on the offspring.

WALTER G. KLUGH, M.D., Hot Springs National Park, Ark.

ANSWER.—The problems and underlying reasons for the excessive use of narcotics and alcohol are the same. It is an escape mechanism from life's conflicts. Chronic morphinism has no true demonstrable pathologic picture. The nature of the disorder is mostly psychologic and not physiologic or pathologic.

Drug addicts can be divided into three groups: 1. The accidental addict, about 10 per cent, composed of normal individuals who develop the habit after the narcotic had been administered for the relief of pain for a period long enough to develop true withdrawal symptoms and who, when the narcotic is discontinued, do not resort to it again, after a successful denarcotization treatment. 2. The recidivists, about 30 per cent, composed of potential narcotic addicts with underlying nervous instability who, after once experiencing narcotic relief and sensation, are unwilling or think they are unable to stand the mental or physical distress which normal persons accept without demanding narcotic relief. 3. The psychopathic personalities, about 60 per cent, recruited from the large class of inadequates with serious underlying defects of personality who find the drug a panacea for their lack of ability to adjust themselves to life's conflicts; the latter group includes the ne'er-do-well type made up of life's misfits who, as a rule, in spite of the exaggerated and sensational news and police reports, are not vicious or dangerous criminals. Addicts make poor criminals. They have no will power, their judgment is defective, they cannot be trusted and they commit, as a rule, only petty crimes and are ostracized and looked down on by the more capable and dangerous criminals.

There is no evidence that the excessive use of morphine affects the germ plasm of ova or spermatozoa or that the offspring are injured by morphinism in the father. However, where the nursing mother is an addict, cases have been reported that when feeding at the breast was discontinued the baby exhibited withdrawal symptoms which were promptly relieved by an opiate.

The danger lies in the descendant's inheriting the unstable nervous system and personality defect of the parent rather than the effect of chronic morphinism.

ESTROGENIC SUBSTANCE FOR ACNE AND FACIAL HYPERTRICHOSIS

To the Editor:—Somewhere I have read that acne vulgaris and facial hypertrichosis have been successfully treated with folliculin ointment. Please explain how it works and where one may procure the ointment?

M.D., Ohio.

ANSWER.—The theory of the use of estrogenic substance for the treatment of acne and hypertrichosis is that both these disorders are due to lack of the estrogenic substance. This is no doubt true for some cases of hypertrichosis and may be part of the basis of some cases of acne vulgaris. Most dermatologists are willing to state that some hormone disturbance is behind dyscrasia of the sebaceous glands which results in acne, but what this disturbance is they are not able to say. Theodore Rosenthal and Raphael Kurzrok (Excretion of Estrin in Acne, *Proc. Soc. Exper. Biol. & Med.* **30**:1150 [May] 1933) found the estrogenic principle regularly present in the urine of normal women but absent from the urine of twenty-seven of the thirty-three women with acne vulgaris whom they tested.

Practical application of this theory to the treatment of acne vulgaris has not been conspicuously successful.

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Michael, J. C.: Observations on the Treatment of Acne Vulgaris, THE JOURNAL, Aug. 3, 1935, p. 327.

McCarthy, Lee, and Hunter, O. B.: Failure of Therapy with Glandular Preparations in Acne Vulgaris, Arch. Dermat. & Syph. **35**: 211 (Feb.) 1937.

Templeton, H. J., and Truman, S. R.: Endocrine Therapy in Acne Vulgaris, *California & West. Med.* **48**: 337 (May) 1938.

In his discussion on the paper of Van Studdiford, C. Guy Lane cited a case of acne occurring at menstrual periods which had resisted x-rays but cleared up when amniotin was given for about seven months and remained clear without further medication. Earlier attempts to stop treatment had resulted in recurrence.

The results of the treatment of hypertrichosis in women is also unpredictable. In most cases no benefit is seen. Once in a while success is achieved for a while, but seldom is the result permanent. The chance of obtaining relief from the excessive growth of hair is much better when other symptoms of ovarian disturbance accompany the hypertrichosis.

While it seems that the use of estrogenic substance is justified in women with some signs of ovarian disturbance accompanied by hypertrichosis or acne vulgaris, especially the form which recurs at the menstrual period and clears up between the periods, it is certainly unwise to use such preparations indiscriminately. In view of the effect in young male rats, its use in boys with acne should be avoided. It is a drug with great and not fully explored potentialities. Its administration in ointment form has the disadvantage of indefinite dosage.

HYPERKERATOSES OF FEET

To the Editor:—A woman aged 24, whose physical examination is otherwise negative, has had thick callosities of the soles, heels and toes of both feet for six years. A surgeon and a dermatologist in consultation agree that this is apparently a simple callosity. Various ointments, including cod liver oil and allantoin, as well as paring down, have been followed by prompt recurrence even when the patient was kept in bed for a month. Roentgen therapy (twelve treatments, details lacking) in a large New York hospital to the extent of producing a slough was otherwise ineffectual. Ultraviolet has been tried. The patient has been saturated with vitamins A, B, C, D and G. The roentgen necrosis is now healed. Please help.

JOSEPH M. GROSS, M.D., Brooklyn.

ANSWER.—Simple calluses are discrete overgrowths of the superficial part of the skin due to pressure or friction rub. Removal of the cause prevents recurrence of the callus. In view of the recurrence with the patient in bed, this would rule out the presence of a simple callus.

Hyperkeratosis, which corresponds to the description in this case, is an overgrowth of the outer layer of the skin which occurs on the plantar surface of the foot.

Hyperkeratosis is frequently a late stage of dermatomycosis. Treatment of the hyperkeratosis in such an instance would be the use of heliotherapy or Whitfield's ointment; 5 per cent salicylic acid in hydrous wool fat may be rubbed vigorously into the skin. Walking barefooted on dry sand is helpful if this is practical.

In addition, there is a hyperkeratotic condition of the skin which occurs in certain persons without known cause. This type may be congenital. Inquiry should be made to ascertain whether arsenic has been used as medication, since this is sometimes a cause of hyperkeratosis. Roentgen treatment has been used in some cases with benefit, but most of these patients are best treated by thoroughly washing the foot with tincture of green soap, which softens the skin. This is followed by an application of 5 per cent salicylic acid in hydrous wool fat, which is rubbed vigorously into the skin several times a day.

HEMORRHAGE AND LEUKEMIA

To the Editor:—In a case of lymphatic leukemia, with the patient in fair condition and of plethoric type, what would be the effect of removal of a large quantity of blood (blood letting)? How long is the time of blood restoration? Which constituents of the blood are first restored? Are the erythrocytes or leukocytes first regenerated? Has this been given any scientific study as to extent or location of faulty regenerative function of the hematopoietic cells?

A. G. LEE, M.D., Texarkana, Ark.

ANSWER.—Although there are no data available on the effect of venesection in leukemia, large spontaneous hemorrhages occur which produce the same effect. Following such an occurrence, red cell and hemoglobin regeneration is slow—often there is no regeneration, especially in the acute forms of the disease. This poor response is probably due to the fact that the red cell forming tissue is already overtaxed. In several observed cases extensive hemorrhage did not affect the white cells appreciably.

No literature is available on the use of venesection as a test in leukemia. However, the cause of the anemia in leukemia is not settled, being variously thought to be due to malfunction of the marrow or to increased peripheral destruction.

POISONING FROM SELENIUM

To the Editor:—Kindly give me a brief description of so-called poisoning by selenium fumes and the treatment.

M.D., New York.

ANSWER.—Acute or chronic poisoning by selenium may be produced by inhalation of volatile selenium compounds or seleniferous dusts. In animals inhalation of moderately high concentrations of hydrogen selenide (H_2Se) produced fatty metamorphosis of the liver, accompanied later by hypertrophy of the spleen. At lower concentrations of hydrogen selenide, animals show less marked liver and spleen damage. Numerous studies have shown the toxic properties of selenium on ingestion or injection of its compounds. In general the site of greatest injury from absorption of selenium compounds is the liver, spleen and kidneys.

In man the symptoms following prolonged absorption of selenium may include pallor, gastrointestinal disturbances, garlic odor of the breath and perspiration, irritation of the nose and throat (rose cold), coating of the tongue, and metallic taste in the mouth. Selenium in the urine is conclusive evidence that absorption of selenium has occurred. Analysis of a morning specimen of urine for selenium is suggested as a diagnostic procedure.

To prevent further absorption of selenium is the first step in the treatment of chronic selenium poisoning. The treatment is symptomatic; some authors suggest the use of a high carbohydrate diet, with increased water intake, as excretion is predominantly renal.

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TUBERCULOSIS AND CAISSON DISEASE

To the Editor:—A man aged 38 suffered an attack of caisson disease in March 1936, from which he made an almost complete recovery. Examination of his lungs at that time did not reveal any pathologic condition and no x-ray examination was made. In March 1937 a diagnosis of active pulmonary tuberculosis (positive sputum and roentgenograms) was made. He began to cough about a month before the roentgenograms were taken. Is there any possible connection between the tuberculosis and the caisson disease? Is there any literature on this subject?

M.D., New Jersey.

ANSWER.—There is no logical explanation of a relationship between the attack of caisson disease which the patient suffered in March 1936 and the presence of active pulmonary tuberculosis in March 1937. As pulmonary tuberculosis frequently is not detected by physical examination and since x-ray films were not made, it is possible that pulmonary disease was present in a presymptom stage at the time he had caisson disease. Often chronic pulmonary tuberculosis can be located by x-ray films several years before significant abnormal physical signs or symptoms make their appearance. Therefore it is possible that x-ray examination in March 1936 would have revealed evidence of the disease.

On the other hand, it is possible that tuberculosis was not present in March 1936 and that the patient became infected subsequently to that date, since many persons now contract their first infections in adult life. No mention is made of a tuberculin test having been administered at the time of or subsequent to the attack of caisson disease. If this was done, and the patient was a nonreactor at that time, there is excellent evidence that the tuberculosis is of recent origin. Although the time is short for the development of chronic pulmonary tuberculosis from a recent infection there is occasionally such an occurrence. However, with the evidence presented there would not appear to be any association between caisson disease and tuberculosis.

RABIES PROPHYLAXIS

To the Editor:—A recent outbreak of rabies resulted in scores of people presenting themselves for preventive treatment. Their contacts varied from ordinary petting of rabid animals to actual bites. One department in the area gave treatment to every one who had had any contact at all, whereas the other department required an actual bite of more than trivial extent before Pasteur treatment was started. Must a skin wound from the bite of a rabid animal be of subcutaneous depth or more before Pasteur treatment is indicated? Do small, superficial breaks in the skin indicate treatment if the patient has had contact with a rabid animal?

JOHN M. COLLINS, M.D., Seattle.

ANSWER.—In the treatment of a person who may have been bitten by a rabid animal, the guiding principle is always safety first. Take no chances! The answer to the first question is No. Any wound made by the bite of a rabid animal, no matter how insignificant, may admit the infection. The answer to the second question is Yes in case it is possible that the break in the skin was made by the bite of a rabid animal.

COPPER IN DRINKING WATER

To the Editor:—Will you please advise me as to whether or not any toxic effects could result from using drinking water which contains 0.65 part of copper per million parts of water?

ARTHUR H. LONDON JR., M.D., Durham, N. C.

ANSWER.—The United States Treasury Department standard for water supplies to be furnished for drinking and culinary purposes by interstate carriers requires that the copper content of the water shall not exceed 0.2 part per million. In recent years there has been some criticism of this standard by biochemists and physiologists, claiming that it is too low and that harmful effects may not result if the copper content is as high as 1 part per million. Experience with the physiologic effects of copper in the light of its more liberal use in water service connections and for control of algae in the public water supplies seems to indicate that the official standard is quite conservative.

GLASSES FOR PROTECTION AGAINST ULTRAVIOLET RAYS FROM A QUARTZ BURNER

To the Editor:—Do ordinary glass or polaroid glasses give adequate protection against ultraviolet rays directed into the face with the eyes open from a Hanovia mercury vapor quartz burner? What is the lens in the goggle furnished by the manufacturer? The representative is unable to tell me.

M.D., New York.

ANSWER.—Ordinary glass and polaroid glasses do give adequate protection against ultraviolet rays directed into the face with the eyes open with any quartz burner emitting ultraviolet rays. The polaroid glasses, however, are rather expensive and there is no point in using them for protection against ultraviolet rays, as ordinary winlow glass serves the same purpose.

Glass in goggles ordinarily used when taking ultraviolet radiation treatments are of various styles and usually are colored merely to soften the glare of the light. There are glass preparations on the market for protection against the effects of screening out infra-red radiation as well as ultraviolet radiation.

CHILDBIRTH AND OBESITY

To the Editor:—What would be the proper procedure for delivery in the following case? A negro woman 29 years old and weighing 325 pounds (147 Kg.) had one child after a difficult labor sixteen years ago and another stillborn child nine years ago. At both previous pregnancies her weight was under 200 pounds. She is at term. The question has arisen whether this woman should be delivered by cesarean section without previous test of labor or should be allowed to go into labor and the case handled from then on according to indication.

M.D., Indiana.

ANSWER.—A cesarean section in a patient weighing 325 pounds is a formidable procedure. It necessarily carries with it a considerable risk for the mother. The query does not state that the previous stillborn children were the result of difficult labors. The labors may not have contributed to the fetal deaths. Unless some unusual complication arises, the safest procedure is to allow the patient to go into labor and deliver her through the birth canal. The rapid increase in her weight need not necessarily contribute to a more difficult natural delivery.

ORAL IRON MEDICATION AND CONSTIPATION

To the Editor:—What method or methods may be used to prevent the constipation so frequently caused by oral iron medication?

E. N. MURRAY, M.D., Camden, N. J.

ANSWER.—Oral iron medication usually causes constipation in persons who already have such a tendency. Sometimes the constipating effect seems to be worse when small doses are given. Ferrous sulfate and iron and ammonium citrates given on an empty stomach between meals in 0.2 Gm. (3 grain) and 2 Gm. (30 grain) doses three times a day respectively are frequently less costive than other preparations. If this fails, the usual treatments for constipation must be tried.

SUPERNUMERARY FINGERS AND TOES

To the Editor:—Please state what you consider the best time in life to operate on supernumerary fingers and toes.

M.D., Ontario.

ANSWER.—In general it would be wise to remove supernumerary fingers and toes early in life, in fact as soon as the parts involved are well enough developed to make an accurate surgical removal possible after a careful study of the roentgenograms. Sometimes these extra digits are attached by separate joints, sometimes one joint suffices for both, and therefore the amputation must avoid impairment of the function of the adjacent finger or toe. In general this type of plastic surgery should be done about the end of the first year.

VAGINAL ABSORPTION

To the Editor:—On page 173 of the January 14 issue of THE JOURNAL the question "Is there any absorption from the vaginal mucosa?" is answered "There is no absorption from the intact vaginal mucosa. There can be absorption from a damaged mucosa. It is not probable that absorption in such a manner occurs often enough to be of practical importance." Absorption of the estrogens from the vaginal mucosa is well known. Serious poisoning and fatalities have been all too frequently reported after the use of mercury bichloride for vaginal douches. In addition there is the study by Macht (*J. Pharmacol. & Exper. Therap.* **10**: 509 [Jan.] 1918) in which it is stated in the conclusions: "It has been shown that a large number of drugs and poisons . . . alkaloids, inorganic salts, esters and antiseptics can be and are easily absorbed through the vaginal wall."

WALTER MODELL, M.D., New York.

Medical Examinations and Licensure

COMING EXAMINATIONS

STATE AND TERRITORIAL BOARDS

ALABAMA: Montgomery, June 20-22. Sec., Dr. J. N. Baker, 517 Dexter Ave., Montgomery.

ARKANSAS: *Medical (Regular).* Little Rock, June 8-9. Sec., State Medical Board of the Arkansas Medical Society, Dr. L. J. Kosminsky, 317 State Line, Texarkana. *Medical (Eclectic).* Little Rock, June 8-9. Sec., Dr. Clarence H. Young, 1415 Main St., Little Rock. *Basic Science.* Little Rock, May 22. Sec., Mr. Louis E. Gebauer, 701 Main St., Little Rock.

CALIFORNIA: *Written examinations.* San Francisco, July 10-13, Los Angeles, Aug. 7-10, and Sacramento, Oct. 16-19. *Oral examinations* (required when reciprocity application is based on a state certificate or license issued ten or more years before filing application in California), Los Angeles, August 7, and San Francisco, Nov. 15. Sec., Dr. Charles B. Pinkham, 420 State Office Bldg., Sacramento.

CONNECTICUT: *Basic Science.* New Haven, June 10. *Prerequisite to license examination.* Address State Board of Healing Arts, 1895 Yale Station, New Haven. *Medical (Regular).* Hartford, July 11-12. *Endorsement.* Hartford, July 25. Sec., Dr. Thomas P. Murdock, 147 W. Main St., Meriden. *Medical (Homeopathic).* Derby, July 11-12. Sec., Dr. Joseph H. Evans, 1488 Chapel St., New Haven.

DELAWARE: Dover, July 11-13. Sec., Medical Council of Delaware, Dr. Joseph S. McDaniel, 229 S. State St., Dover.

DISTRICT OF COLUMBIA: *Basic Science.* Washington, June 26-27. *Medical.* Washington, July 10-11. Sec., Commission on Licensure, Dr. George C. Ruhland, 203 District Bldg., Washington.

FLORIDA: Jacksonville, June 19-20. Sec., Dr. William M. Rowlett, Box 786, Tampa.

GEORGIA: Atlanta, June 7-8. Joint-Sec., State Examining Boards, Mr. R. C. Coleman, 111 State Capitol, Atlanta.

ILLINOIS: Chicago, June 20-22 and Oct. 17-19. Superintendent of Registration, Department of Registration and Education, Mr. Homer J. Byrd, Springfield.

INDIANA: Indianapolis, June 20-22. Sec., Board of Medical Registration and Examination, Dr. J. W. Bowers, 301 State House, Indianapolis.

IOWA: Iowa City, June 6-8. Dir., Division of Licensure and Registration, Mr. H. W. Grefe, State Department of Health, Capitol Bldg., Des Moines.

KANSAS: Kansas City, June 13-14. Sec., Board of Medical Registration and Examination, Dr. J. F. Hassig, 905 N. 7th St., Kansas City.

KENTUCKY: Louisville, June 7-9. Sec., State Board of Health, Dr. A. T. McCormack, 620 S. Third St., Louisville.

MAINE: Augusta, July 11-12. Sec., Board of Registration of Medicine, Dr. Adam P. Leighton, 192 State St., Portland.

MARYLAND: *Medical (Regular).* Baltimore, June 20-23. Sec., Dr. John T. O'Mara, 1215 Cathedral St., Baltimore. *Medical (Homeopathic).* Baltimore, June 20-21. Sec., Dr. John A. Evans, 612 W. 40th St., Baltimore.

MASSACHUSETTS: Boston, July 11-13. Sec., Board of Registration in Medicine, Dr. Stephen Rushmore, 413-F State House, Boston.

MICHIGAN: Ann Arbor and Detroit, June 14-16. Sec., Board of Registration in Medicine, Dr. J. Earl McIntyre, 100 W. Allegan St., Lansing.

MISSISSIPPI: Jackson, June 21-22. Asst. Sec., State Board of Health, Dr. R. N. Whitfield, Jackson.

MISSOURI: St. Louis, June 1-3. Sec., State Board of Health, Dr. Harry F. Parker, State Capitol Bldg., Jefferson City.

NEBRASKA: Omaha, June 8-9. Dir., Bureau of Examining Boards, Mrs. Clark Perkins, State House, Lincoln.

NEW HAMPSHIRE: Concord, Sept. 14-15. Sec., Board of Registration in Medicine, Dr. T. P. Burroughs, State House, Concord.

NEW JERSEY: Trenton. June 20-21. Sec., Dr. Earl S. Hallinger, 28 W. State St., Trenton.

NEW YORK: Albany, Buffalo, New York, and Syracuse, June 26-29. Chief, Bureau of Professional Examinations, Mr. Herbert J. Hamilton, 315 Education Building, State Education Department, Albany.

NORTH CAROLINA: Raleigh, June 19. Sec., Dr. William D. James, The Hamlet Hospital, Hamlet.

NORTH DAKOTA: Grand Forks, July 5-8. Sec., Dr. G. M. Williamson, 4½ S. Third St., Grand Forks.

OHIO: Columbus, June 6-9. Sec., State Medical Board, Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA: *Basic Science.* Oklahoma City, May 15. Sec. of State, Hon. C. C. Childress, State Capitol, Oklahoma City. *Medical.* Oklahoma City, June 14. Sec., Dr. James D. Osborn, Jr., Frederick.

OREGON: *Medical.* Portland, June 20-22. Sec., Dr. Joseph F. Wood, 509 Selling Bldg., Portland. *Basic Science.* Corvallis, July 8, and Portland, Oct. 28. Sec., State Board of Higher Education, Mr. Charles D. Byrne, University of Oregon, Eugene.

PENNSYLVANIA: Philadelphia and Pittsburgh, July. Sec., Board of Medical Education and Licensure, Dr. James A. Newpher, 400 Education Bldg., Harrisburg.

SOUTH CAROLINA: Columbia, June 27. Sec., Dr. A. Earle Boozer, 505 Saluda Ave., Columbia.

SOUTH DAKOTA: Rapid City, July 18-19. Director, Medical Licensure, Dr. G. J. Van Heuvelen, State Board of Health, Pierre.

TENNESSEE: Memphis, June 15-16. Sec., Dr. H. W. Qualls, 130 Madison Ave., Memphis.

TEXAS: Austin, June 21-23. Sec., Dr. T. J. Crowe, 918 Mercantile Bldg., Dallas.

UTAH: Salt Lake City, June 26-28. Dir., Department of Registration, Mr. G. V. Billings, 326 State Capitol Bldg., Salt Lake City.

VERMONT: Burlington, June 14-16. Sec., Board of Medical Registration, Dr. W. Scott Nay, Underhill.

VIRGINIA: Richmond, June 21-23. Sec., Dr. J. W. Preston, 30½ Franklin Road, Roanoke.

WEST VIRGINIA: Bluefield, July 5. Sec., Public Health Council, Dr. Arthur E. McClue, State Capitol, Charleston.

WISCONSIN: *Basic Science.* Milwaukee, June 3. Sec., Prof. Robert N. Bauer, 3414 W. Wisconsin Ave., Milwaukee. *Medical.* Milwaukee, June 27-30. Sec., Dr. Henry J. Gramling, 2203 S. Layton, Blvd., Milwaukee.

WYOMING: Cheyenne, May 31. Sec., Dr. G. M. Anderson, Capitol Bldg., Cheyenne.

NATIONAL BOARD OF MEDICAL EXAMINERS
SPECIAL BOARDS

Examinations of the National Board of Medical Examiners and Special Boards were published in THE JOURNAL, May 6, page 1857.

Colorado January Report

Dr. Harvey W. Snyder, secretary, Colorado State Board of Medical Examiners, reports the written examination held at Denver, Jan. 4-6, 1939. The examination covered eight subjects and included 161 questions. An average of 75 per cent was required to pass. Three candidates were examined, all of whom passed. Four physicians were licensed by endorsement on January 3. The following schools were represented:

School	PASSED	Year Grad.	Per Cent
Northwestern University Medical School		(1938)	89
Osteopaths *			82, 86

School	LICENSED BY ENDORSEMENT	Year Grad.	Endorsement of
Rush Medical College		(1937)	N. B. M. Ex.
State University of Iowa College of Medicine		(1926)	Iowa
St. Louis University School of Medicine		(1936)	Illinois
Baylor University College of Medicine		(1926)	Texas

* Licensed to practice medicine and surgery.

Book Notices

The Treatment of Fractures. By Charles Locke Scudder, A.B., Ph.B., M.D., Consulting Surgeon to the Massachusetts General Hospital, Boston. Eleventh edition. Cloth. Price, $12. Pp. 1,208, with 1,717 illustrations. Philadelphia & London: W. B. Saunders Company, 1938.

The treatment of fractures forms a more important part of surgical practice today than ever before because of much reckless automobile driving and the ever increasing mechanization of industry. In the twelve years since the previous edition of this book there have been many advances in the diagnosis and treatment of fractures, making it necessary for the author to rewrite his material completely. He has deleted all obsolete methods of diagnosis and treatment and added hundreds of new illustrations. Dr. Scudder personally approves the opinions expressed by the twenty other contributors to the volume, men actually engaged in doing the procedures about which they have written. He recognizes the importance of adequate first aid treatment and proper transport, factors which the general practitioner must master. There are forty-seven chapters; however, the size of the book remains convenient. It closes with a chapter on the medicolegal aspects of fractures.

The Essentials of Modern Surgery. Edited by R. M. Handfield-Jones, M.C., M.S., F.R.C.S., Surgeon to Out-Patients, St. Mary's Hospital, London, and A. E. Porritt, M.A., M.Ch., F.R.C.S., Surgeon to His Majesty's Household. Cloth. Price, $9. Pp. 1,126, with 501 illustrations. Baltimore: William Wood & Company, 1938.

This book succeeds in its attempt to present the essentials of modern surgery in concise form for both students and practitioners. While it represents the work of fifteen authors, who are responsible for individual chapters, the chapters contributed by the senior author are especially fine. The numerous and instructive illustrations are unsurpassed in any similar volume. In arrangement the book resembles many current volumes in the same field, beginning with an entirely adequate treatment of the pathology of inflammation, infections and the principles of surgical technic and then passing on to regional surgery. The text is lucid, interesting and in general authoritative. However, an American reviewer is surprised to find in the chapter on anesthesia considerable discussion of chloroform but no mention of ethylene or of some of the more recent inhalation anesthetic agents. A more adequate discussion of diseases of the biliary tract and of cholecystography seems desirable. No mention is made of the use of the gastroscope and its place in the diagnosis of gastric lesions. More serious is the omission of any discussion of the problems of water balance, dehydration, and demineralization in preoperative and postoperative treatment and the significance of these factors in acute intestinal obstruction, acute dilatation of the stomach and high intestinal fistulas. These are minor criticisms, however, and both the authors and the publisher are to be highly commended for the production of this book.

Clinical and Experimental Investigations in Agranulocytosis with Special Reference to the Etiology. By Preben Plum. Denne Afhandling er af det lægevidenskabelige Fakultet antaget til offentlig at forsvares for den medicinske Doktorgrad, København. Paper. Pp. 410, with 125 illustrations. London: H. K. Lewis & Co., Ltd.; Copenhagen: NYT Nordisk Forlag, Arnold Busck, 1937.

Most of this volume is devoted to the etiology of granulocytopenia. It is the most comprehensive and accurate compilation of data concerning that disease which has yet appeared. There are twelve chapters, a section of photomicrographs and records of 114 cases of agranulocytosis and other leukopenic diseases, all of which were observed in Denmark by the author or his colleagues. These chapters are followed by a list of drugs incriminated in the production of the disease, a summary, a summary in Danish, and finally a bibliography of 422 references incorporating the literature of the world. As stated in the introduction, the author compiled this work under a fellowship from a Danish fund for medical research and was aided by financial grants from other scientific agencies of Denmark. Therefore it can be considered to be accurate and scientific.

The first chapter considers the history of the disease, and early sporadic cases are cited. Plum concludes that the disease probably did not exist before the report of Schultz in 1922. There is then a thorough discussion of the terminology, geographic distribution, seasonal incidence, sex and age incidence and occupational frequency. Just as in the United States, the disease in Denmark has occurred more frequently in physicians, nurses and others closely associated with the medical profession. In about fifty pages there is an excellent summing up of the clinical symptoms, hematologic signs, pathologic changes and results of laboratory studies. Data from the world literature are presented, and the reports of the ninety cases studied in Denmark are included. Nearly eighty pages is then devoted to the etiology of agranulocytosis, and in this section is found the most interesting material. The author considers the various etiologic concepts that have been presented, including the role of infections, the various predisposing factors, the influence of the menstrual cycle, the role of allergy, the effects of such agents as arsphenamine, benzene, arsenic, bismuth and gold preparations and, finally, the role of aminopyrine. There is then presented an accurate and chronologic record of the development of the theory of causation by drugs, beginning with the report of Kracke in the United States, who is given priority for the development of the concept. The author summarizes the cases reported of granulocytopenia occurring after the administration of aminopyrine, including experimental work with both human beings and animals, and includes his own extensive experiments. There is a brief discussion of the role of other drugs, including the barbituric acid preparations, novaldin, antipyrine, acetanilid, acetophenetidin, dinitrophenol, cinchophen, quinine, neostibosan and sulfanilamide. He states that since the probable etiologic significance of aminopyrine was recognized in Denmark the consumption of the drug has markedly decreased and that there has been a remarkable parallel between the incidence of the disease and the consumption of the drug to the extent that in 1936 the disease had practically disappeared. He concludes that it may be safely assumed that, as far as Denmark is concerned, aminopyrine is in all probability the main etiologic factor in agranulocytosis. He then discusses the pathogenesis, emphasizing the belief that the clinical symptoms are preceded by the hematologic changes and that the infectious processes result from loss of cellular resistance. The final four chapters deal with the diagnosis, differential diagnosis, course, prognosis and treatment. He discusses the merits of treatment with nucleic acid products including pentnucleotide, liver extract, transfusions, x-rays and other measures. A note of pessimism is sounded, for "according to our present knowledge of the etiology of agranulocystosis the therapeutic methods generally employed do not appear particularly expedient" and "the rational thing is to direct our therapeutic efforts against the provoking agent." He stresses the value of

prophylaxis and emphasizes that the most valuable measure in the control of the disease is to reduce the consumption of aminopyrine.

The twenty-six colored illustrations are excellent photomicrographic reproductions of the various mature and immature blood cells, and the black and white ones are mainly of the bone marrow in agranulocytosis. This work is available in both English and Danish. It has been executed with meticulous care and with a high degree of accuracy by one who obviously is a careful worker. It represents an authoritative source of material for all those interested in agranulocytosis and other leukopenic diseases.

Proceedings of the Fourth Conference on Psychiatric Education Undergraduate Instruction, Baltimore, Maryland, April 8-10, 1936. Paper. Pp. 345. New York City: The National Committee for Mental Hygiene, Inc., Division of Psychiatric Education, 1938.

In April 1936 the Fourth Conference on Psychiatric Education was held in Baltimore with Adolph Meyer in the chair. The present volume is a report of the proceedings of this conference and is an extremely comprehensive survey of the subject of psychiatric education. Many reports are included which discuss how much of the medical school schedule is devoted in various universities at the present time to the presenting of psychiatric material to the student. Papers were read in which the ideal curriculum in psychiatry for medical students was considered, and such features as the need of premedical psychology courses and the relationship of other parts of the medical curriculum to psychiatry were discussed in some detail. Considerable attention was devoted to the matter of how psychiatry should be taught, how the class work should be divided, the purpose of ward walks and the need for special training in the teaching of psychiatry. About a third of these proceedings is allotted to an appendix, which deals with the psychiatric course presented at the Johns Hopkins Hospital. There are classifications of mental disease along the lines of those in use at the Johns Hopkins University School of Medicine, and forms for history taking and recording are also shown. Also included in the appendix are several papers, chiefly by Dr. Meyer, which were not read at this conference but which treat of various aspects of psychiatry.

Traité de chirurgie orthopédique. Publié sous la direction de L. Ombrédanne et P. Mathieu. Secrétaires de la rédaction: M. Lance, G. Huc et P. Padovani. Tome I. Physiologie, pathologie et therapeutique. Cloth. Price, 300 francs. Pp. 902, with 433 illustrations. Tome II. Physiologie, pathologie et therapeutic générales (fin.) Rachis; membre supérieur. Half-cloth. Pp. 903-1921, with illustrations. Tome III. Deuxième partie: Rachis; membre supérieur (suite et fin). Cloth. Price, 300 francs. Pp. 1919-2875, with illustrations. Tome IV. Troisième partie: Bassin; membre inférieur. Half-cloth. Price, 300 francs. Pp. 2873-3778, with illustrations. Tome V. Troisième partie: Rachis; membre inférieur (suite et fin). Half-cloth. Price, 300 francs. Pp. 3777-4482, with illustrations. Paris: Masson & Cie, 1937.

This set of five volumes is the finest material on orthopedic surgery that has come from France. It reflects the knowledge of French orthopedic surgeons and general surgeons interested in bone and joint and related lesions. The contributors were chosen with great care, and they have done themselves, both individually and collectively, great honor. Some of the most notable are Ombredanne, Mathieu, Lance, Huc, Padovani, Calvé, Delahaye, Ducroquet, Fontaine, Ferdet, Inglerans, Lamy, Lasserre, LeDoux-Lebard, LeFort, Leriche, Leveuf, Mouchet, Nové-Josserand, Policard, Ponzet, Rendu, Rocher, Roederer, Sorrel, Sorrel-Dejerine and Tavernier. Volume I treats congenital malformation of the extremities, the skeletal system, lesions of bones, diseases of joint and surgical procedures on joints, diseases of muscles, tendons and aponeuroses, and lesions of the skin. Volume II deals with diseases of the nervous system, including spastic paralysis, infantile paralysis and wounds of nerves, as well as lesions of the blood vessels of interest to the orthopedic surgeon, diseases of the blood, diagnostic roentgenography in orthepedic surgery, apparatus, including splints and braces, plaster of paris and celluloid, treatment in general of wounds, kinesitherapy, physical therapy and climatotherapy. The second part of this volume discusses the spine and all its lesions. Volume III contains a discussion of tumors of the vertebra, unusual diseases, the shoulder girdle and all its lesions, the thorax, the scapula, the shoulder, the arm, the elbow, the forearm, the wrist and the hand. Volume IV deals with the pelvis, the hip, the thigh and the knee from the standpoint of congenital defects and infectious diseases. Volume V deals with the leg, the ankle joint and the foot, amputations, apparatus of all types, paralysis of the upper extremities, operative technic and apparatus for paralysis of the upper extremities and for the lower extremities, and the procedures for equalization of the length of the lower extremities. The text, composition and illustrations are excellent. The collaborators are unfamiliar with or unwilling to accept the work of surgeons in other countries. There is an occasional reference to Italian and German literature but few to that of the English speaking countries. The work is encyclopedic; it cannot be recommended to the individual English reading orthopedic surgeon but is a valuable reference work.

Archiv und Atlas der normalen und pathologischen Anatomie in typischen Röntgenbildern. Röntgendiagnostik der Gelenke mittels Doppelkontrastmethode. Von Dr. J. Oberholzer, Chefarzt am Kantonalen Krankenhaus Wallenstadt. Mit Vorwort von Dr. med. E. Bircher, A. Spitaldirektor Aarau. Fortschritte auf dem Gebiete der Röntgenstrahlen, Ergänzungsband LVI. Herausgeber: Prof. Grashey. Paper. Price, 23 marks. Pp. 128, with 134 illustrations. Leipzig: Georg Thieme, 1938.

The author discusses diagnostic roentgenography of joints by means of a double contrast method. The technic of the injection of various joints by the contrast method and the x-ray technic are effectively demonstrated. The discussions and illustrations, especially of the knee joint, are excellent and offer a fine method of precise diagnosis of interesting and often difficult problems.

Analysis of Parergasia. By Gladys C. Terry, Research Associate, Neurological Institute of New York, Columbia University, New York, and Thomas A. C. Rennie, M.D., Associate in Psychiatry, Phipps Psychiatric Clinic, Johns Hopkins University, Baltimore. With an introduction by Adolf Meyer, Henry Phipps Professor of Psychiatry, Johns Hopkins University, Baltimore. Nervous and Mental Disease Monograph Series No. 64. Boards. Price, $4. Pp. 202. New York: Nervous and Mental Disease Publishing Company, 1938.

Seventy-seven cases of parergasia or schizophrenia from the basis for the present volume. A mass of data is arranged according to the life experience chart of Adolph Meyer. A tremendous amount of material is presented and yet a purely descriptive use is made of it. The parergastic subject's life defeat is based on poor constitution and personality traits which cause failure in adaptation to life situations. However, to accept life situations which impinge on the individual even in the early years as dynamic factors is the great defect of the meyerian psychobiology. It gives, in other words, lip service to the term dynamic and treats it purely descriptively. The material is so organized in a summarized descriptive form that no truly dynamic reconstruction could be achieved. Life experiences are stressed as important to the individual's fate yet their meaning in terms of the individual is ignored. The earliest infantile situations are virtually overlooked and the patient is considered as crushed by relatively late experiences impinging on a psychobiologic unit poorly prepared for them. What makes him unable to live through the multifarious experiences which are not so specific for his life? In this regard the followers of Meyer ignore the earliest infantile experiences which in their effects on the personality may cause truly dynamic alterations and prepare the way for the future failure in adjustment. These earliest interactions are so completely overlooked in this book that a tremendous amount of work becomes valuable only in a descriptive sense.

The Functions of Human Voluntary Muscles. By Norman D. Royle, M.D., Ch.M., F.R.A.C.S., Senior Orthopaedic Surgeon, Lewisham Hospital, Sydney. Cloth. Price, 3s. 6d. Pp. 42, with 11 illustrations. Sydney & London: Angus & Robertson Limited, 1938.

Dr. Royle developed a remarkable control of the individual muscles of his own body, thus acquiring a unique knowledge of the actions of certain muscles. This knowledge was the basis of a series of lectures in the department of anatomy at the University of Sydney, Australia, which in turn were the basis of this book. He discusses the movements of the vertebral column, jaws, shoulder girdle, upper limbs and lower limbs and emphasizes the importance of a knowledge of the formation as well as of the attachments and nerve supply of a muscle in determining appropriate treatment. Various methods of approach in ascertaining the formation of muscles, namely

anatomic, pathologic, electrical and physiologic methods, are described, and the principal or prime mover, reverse action, fixation muscles and synergic action are discussed. At first thought this book seems elementary, but it is well worth the time of any physician and especially the orthopedic surgeon.

Röntgendiagnostik der Knochen- und Gelenkkrankheiten. Von Professor Dr. Robert Kienböck. Heft 5 (Abteilung Gelenkkrankheiten): Rheumatoide Gelenkstuberkulose. Paper. Price, 60 marks. Pp. 451-1049, with 610 illustrations. Berlin & Vienna: Urban & Schwarzenberg, 1938.

This elaborate and comprehensive book on rheumatoid tuberculosis of the joints, volume 5 in a series on the x-ray diagnosis of bone and joint diseases, is divided into chapters on tuberculosis of the joints of the hand, the shoulder, the foot, the knee, the hip, the sacro-iliac region and the vertebrae. The second part deals with multiple lesions, including diseases, Heberden's nodes and mono-articular infections. There are several excellent illustrations of rhizomelic spondylosis with the bamboo pole appearance involving the cervical thoracic and lumbar vertebrae. The author discusses 378 cases and presents an extensive bibliography under eighteen headings. This fine volume is chiefly a reference book, highly technical and of value to those who read German easily.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Workmen's Compensation Acts: Disability from Displacement of Fifth Lumbar Vertebra. — Donker, a man weighing about 200 pounds (90.7 Kg.), in the course of his employment Jan. 3, 1936, fell on his back and could not continue working. His legs felt numb and he had pains in his legs and back. He was taken to a physician in Sioux City for treatment but the pains persisted after several weeks of treatment. The physician then removed the patient's coccyx. The last week in April the physician released the patient, although at the time the patient complained of a burning sensation up and down his spine and numbness in his legs. Another physician, consulted about May 20, prescribed a brace for the lower part of the body and rendered treatment until August 19, but the pains continued to grow worse. On the last date named the workman entered the Veterans' Hospital in Lincoln, Neb., where he was kept in a cast for five weeks and was given a brace, his tonsils were removed and some teeth were extracted. After his release from the hospital, November 3, he instituted proceedings under the workmen's compensation act of Nebraska. At the trial he testified that he had very little use of his legs and was forced to wear the brace continuously, that his legs were numb and that he suffered a burning sensation of the spine and hip pains. The trial court awarded compensation for temporary total disability and the employer and his insurance carrier appealed to the Supreme Court of Nebraska.

The only questions for determination by the Supreme Court were the extent of the injuries suffered by the workman and the nature of the resulting disabilities. A physician who personally supervised the treatment given the workman at the Veterans' Hospital and who examined him during the month preceding the trial testified that in his opinion Donker was permanently and totally disabled. As to the condition of the lower portion of Donker's spine, there was expert testimony that the fifth lumbar vertebra was displaced forward so as to be no longer in line with the sacrum and that the space between the two was wider than normal. That condition, the witness testified, might have been present before the injury, might have been aggravated by the injury or might have been caused entirely by the injury. Donker's family physician, who attended him after his release from the Veterans' Hospital, testified, from the results of examinations made including the use of roentgenograms and his previous knowledge of the patient's condition, that the fall on January 3 would be sufficient to cause the injuries from which he suffered, that in his opinion the workman's disability was due wholly and entirely to the fall and that the workman was totally and permanently disabled.

The Supreme Court concluded that the finding of the trial court that the workman was suffering from at least temporary total disability was clearly established by the evidence. It was undisputed that prior to January 3 Donker was an able bodied laborer, capable of continuously performing hard work without ill effects. After that date his condition changed markedly. The only explanation offered for that change by the defendant was that the workman was a malingerer. But the evidence, the court thought, preponderated against that conclusion.

Taking into consideration all the evidence, the Supreme Court concluded that the judgment of the trial court awarding compensation was justified. The judgment was therefore affirmed. —*Donker v. Central West Public Service Co. (Neb.), 280 N. W. 168.*

Society Proceedings

COMING MEETINGS

American Medical Association, St. Louis, May 15-19. Dr. Olin West, 535 North Dearborn St., Chicago, Secretary.

American Academy of Tuberculosis Physicians, St. Louis, May 13-14. Dr. Arnold Minnig, 638 Metropolitan Bldg., Denver, Secretary.
American Association for the Study of Goiter, Cincinnati, May 22-24. Dr. W. Blair Mosser, 133 Biddle St., Kane, Pa., Secretary.
American Association for Thoracic Surgery, Los Angeles, July 5-7. Dr. Richard H. Meade Jr., 2116 Pine St., Philadelphia, Secretary.
American Association of Genito-Urinary Surgeons, Williamsburg, Va., May 24-26. Dr. Charles C. Higgins, 2050 East 93d St., Cleveland, Secretary.
American Association of Industrial Physicians and Surgeons, Cleveland, June 5-8. Dr. V. S. Cheney, Armour and Company, Union Stock Yards, Chicago, Secretary.
American Bronchoscopic Society, Rye, N. Y., May 26. Dr. Lyman Richards, 319 Longwood Ave., Boston, Secretary.
American College of Chest Physicians, St. Louis, May 13-14. Dr. Robert B. Homan Jr., 109 North Oregon St., El Paso, Texas, Secretary.
American Dermatological Association, Montebello, Canada, May 31-June 3. Dr. Fred D. Weidman, University of Pennsylvania Medical Laboratories, Philadelphia, Secretary.
American Gynecological Society, White Sulphur Springs, W. Va., May 22-24. Dr. Richard W. TeLinde, 11 East Chase St., Baltimore, Secretary.
American Laryngological Association, Rye, N. Y., May 24-26. Dr. James A. Babbitt, 1912 Spruce St., Philadelphia, Secretary.
American Neurological Association, Atlantic City, N. J., June 5-7. Dr. Henry A. Riley, 117 East 72d St., New York, Secretary.
American Ophthalmological Society, Hot Springs, Va., June 5-7. Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn., Secretary.
American Orthopedic Association, Buffalo, N. Y., June 5-8. Dr. Ralph K. Ghormley, 110 Second Ave. S.W., Rochester, Minn., Secretary.
American Otological Society, New York, May 22-23. Dr. Thomas J. Harris, 104 East 40th St., New York, Secretary.
American Proctologic Society, Brooklyn, N. Y., June 25-27. Dr. Curtice Rosser, 710 Medical Arts Bldg., Dallas, Texas, Secretary.
American Radium Society, St. Louis, May 15-16. Dr. Frederick W. O'Brien, 465 Beacon St., Boston, Secretary.
American Rheumatism Association, St. Louis, May 15. Dr. Loring T. Swaim, 372 Marlborough St., Boston, Secretary.
American Society for the Study of Allergy, St. Louis, May 15-16. Dr. J. Harvey Black, 1405 Medical Arts Bldg., Dallas, Texas, Secretary.
American Urological Association, White Sulphur Springs, W. Va., May 29-June 1. Dr. Clyde L. Deming, 789 Howard Ave., New Haven, Conn., Secretary.
Associated Anesthetists of the United States and Canada, St. Louis, May 15. Dr. F. H. McMechan, 318 Hotel Westlake, Rocky River, Ohio, Secretary General.
Association for the Study of Internal Secretions, St. Louis, May 13-14. Dr. E. Kost Shelton, 921 Westwood Blvd., Los Angeles, Secretary.
Connecticut State Medical Society, New Haven, May 25-26. Dr. Creighton Barker, 258 Church St., New Haven, Secretary.
Maine Medical Association, Poland Springs, June 25-27. Dr. F. R. Carter, 22 Arsenal St., Portland, Secretary.
Massachusetts Medical Society, Worcester, June 6-8. Dr. Alexander S. Begg, 8 Fenway, Boston, Secretary.
Medical Library Association, Newark, N. J., June 27-29. Miss Janet Doe, 2 East 103d St., New York, Secretary.
Minnesota State Medical Association, Minneapolis, May 31-June 2. Dr. B. B. Souster, 11 West Summit Ave., St. Paul, Secretary.
Montana, Medical Association of, Butte, June 28-30. Dr. Thomas L. Hawkins, 50 North Main St., Helena, Secretary.
National Gastroenterological Association, New York, June 1-2. Dr. G. Pandolph Manning, 1819 Broadway, New York, Secretary.
National Tuberculosis Association, Boston, June 26-29. Dr. Charles J. Hatfield, 50 West 50th Street, New York, Secretary.
New Hampshire Medical Society, Manchester, June 8-9. Dr. Carleton R. Metcalf, 5 South State St., Concord, Secretary.
New Jersey, Medical Society of, Atlantic City, June 6-8. Dr. Alfred Stahl, 55 Lincoln Park, Newark, Secretary.
Pacific Coast Oto-Ophthalmological Society, San Francisco, June 19-22. Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Secretary.
Pacific Northwest Medical Association, Spokane, Wash., June 26-29. Dr. C. W. Countryman, 407 Riverside Avenue, Spokane, Wash., Secretary.
Rhode Island Medical Society, Providence, June 7-8. Dr. Guy W. Wells, 124 Waterman St., Providence, Secretary.
Society of Surgeons of New Jersey, Elizabeth, May 25. Dr. Walter B. Mount, 21 Plymouth St., Montclair, Secretary.
Vancouver Medical Association Summer School, Vancouver, B. C., June 6-9. Dr. W. W. Simpson, 203 Medical Dental Bldg., Vancouver, B. C., Secretary.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1929 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but may be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

Alabama State Medical Assn. Journal, Montgomery

8: 273-304 (Feb.) 1939

Fallacies in the Conduct of Labor. J. R. Garber, Birmingham.—p. 273.
Diagnosis of Intracranial Tumor. W. A. Smith, Atlanta, Ga.—p. 276.
Diagnosis and Treatment of Pleural Effusions. K. N. Joseph, Birmingham.—p. 281.
Some Clinical Observations in Asthmatic Cases with Methods of Management. J. W. Boggess Jr., Guntersville.—p. 285.

American Heart Journal, St. Louis

17: 131-258 (Feb.) 1939

Reversible Cardiac Enlargement in a Case of Congenital Cavernous Hemangioma. R. Matas and B. R. Heninger, New Orleans.—p. 131.
Calcareous Disease of the Aortic Valve: Study of 228 Cases. T. J. Dry and F. A. Willius, Rochester, Minn.—p. 138.
Evaluation of Heart Volume Determinations by the Rohrer-Kahlstorf Formula as a Clinical Method of Measuring Heart Size. W. J. Comeau and P. D. White, Boston.—p. 158.
*Effects of Alkalosis and of Acidosis on the Human Electrocardiogram. P. S. Barker, Ann Arbor, Mich.; E. L. Shrader and Ethel Ronzoni, St. Louis.—p. 169.
Studies on Mercurial Diuresis: II. Immediate Effect on Venous Blood Pressure. I. F. Volini and R. O. Levitt, Chicago.—p. 187.
Plethysmographic Studies of Peripheral Blood Flow in Man: I. Criteria for Obtaining Accurate Plethysmographic Data. D. I. Abramson, H. Zazeela and J. Marrus, Cincinnati.—p. 194.
Id.: II. Physiologic Factors Affecting Resting Blood Flow in the Extremities. D. I. Abramson, H. Zazeela and J. Marrus, Cincinnati.—p. 206.
Use of Magnesium Sulfate in Measurement of Circulation Time. M. Bernstein and S. Simkins, Philadelphia.—p. 218.

Effects of Alkalosis and Acidosis on Electrocardiogram.—Barker and his co-workers observed pronounced flattening of the T waves in a patient with hysterical overventilation and tetany. This prompted an experimental investigation of the effects of alkalosis and acidosis on forty electrocardiograms of seven human volunteers. Alkalosis produced by voluntary overventilation or by the ingestion of sodium bicarbonate was accompanied by a reduction in the amplitude of the T wave. Acidosis produced by exercise or by the ingestion of ammonium chloride was accompanied by an increase in the amplitude of the T wave. While the clinical application of these observations is probably quite limited, they may offer an explanation for occasional electrocardiographic changes not otherwise understood.

American Journal of Cancer, New York

35: 159-326 (Feb.) 1939. Partial Index

Changes in the Nature of the Stroma in Vagina, Cervix and Uterus of the Mouse Produced by Long-Continued Injections of Estrogen and by Advancing Age. L. Loeb, V. Suntzeff and E. L. Burns, St. Louis.—p. 159.
Relative Potency of Carcinogenic Compounds. J. Iball, London, England.—p. 188.
Effect of Diet on Tumors Induced by Ultraviolet Light. C. A. Baumann and H. P. Rusch, Madison, Wis.—p. 213.
Blood Groups in Relation to the Agglutination of Human Red Blood Corpuscles by Heterologous (Rat) Serums. T. Lumsden, London, England.—p. 222.
Radioactivity of Potassium in Tumor Tissue. A. Lasnitzki, Manchester, England.—p. 225.
Sex as a Factor in the Prognosis of Hodgkin's Disease. E. Epstein, Oakland, Calif.—p. 230.
Sarcoma of Soft Tissues. E. M. Burke, Buffalo.—p. 234.
Multiple Malignant Tumors, with Involvement of the Lower Bowel: Report of Multiple Primary and Contact Growths. H. E. Bacon, Philadelphia.—p. 243.
Tumor-like Hyperplasia of the Interstitial Cells in the Testis: Case. B. Snellman.—p. 258.
A Hopeful Factor in the Cancer Problem. W. C. MacCarty Sr., Rochester, Minn.—p. 275.

American J. Obstetrics and Gynecology, St. Louis

37: 183-362 (Feb.) 1939. Partial Index

Occurrence and Significance of Nucleated Erythrocytes in Fetal Vessels of Placenta. C. T. Javert, New York.—p. 184.
*Effect of Calcium on Uterine Contractions and on Uterine Response to Intravenously Injected Oxytocics. D. N. Danforth and A. C. Ivy, Chicago.—p. 194.
Urologic Complications Following Complete and Supravaginal Hysterectomy: Comparative Study. A. J. Murphy, New York.—p. 201.
Critical Analysis of Blood Loss Following Delivery, with Special Reference to Value of Ergotrate. A. M. Reich, New York.—p. 224.
*Etiologic Factors in Sterility: Report of Study of 150 Cases. J. Kotz and Elizabeth Parker, Washington, D. C.—p. 233.
Neonatal Mortality at the Philadelphia Lying-in Hospital: A Seven Year Study. R. M. Tyson, Philadelphia.—p. 241.
Postpartum Hemorrhage as Cause of Death. C. T. Beecham, Philadelphia.—p. 258.
Inhibiting Effect of Pentavalent Arsenicals on Trichomonas: Comparative Efficacy and Toxicity of Acetarsone, Carbarsone and Aldarsone in Laboratory Studies. A. E. Rakoff, Philadelphia.—p. 265.
Treatment of Pelvic Inflammation by Iontophoresis of a Choline Compound. A. Jacoby, New York.—p. 272.
Comparative Study of Effectiveness of Two Antiseptics in Preventing Infection Following Delivery: Report on 1,000 Cases. J. E. Tritsch, New York.—p. 277.
Use of Blood Transfusions in Treatment of Pyelitis Complicating Pregnancy. L. H. Douglass and K. Laughlin, Baltimore.—p. 282.
Blood Sugar Changes in Labor and After Delivery. A. L. DeCamp, Fayetteville, N. C.—p. 285.
Relation Between Hydatid Moles, Relative Ischemia of the Gravid Uterus and the Placental Origin of Eclampsia. E. W. Page, Berkeley, Calif.—p. 291.
*Bartholinitis and Skeneitis Due to Trichomonas Vaginalis. H. A. Shelanski and S. P. Savitz, Philadelphia.—p. 294.
Tubal Pregnancy with Tuberculous Salpingitis. C. S. Stevenson and L. R. Wharton, Baltimore.—p. 303.
Successful Termination of Pregnancy Following Bilateral Sympathectomy. J. M. Melick, Worcester, Mass.—p. 334.

Effect of Calcium on Uterine Contractions.—Danforth and Ivy find that calcium is necessary for the maintenance of the tone, contractility and coordinated activity of the postpartum uterus of the dog and rabbit. When the available calcium is sufficiently reduced by the use of soluble sodium hexametaphosphate, uterine contractions do not occur. The oxytocic action of pitocin, ergonovine and histamine is potentiated by calcium. Doses of pitocin, ergonovine and histamine, which are normally adequate for a uterine response, are ineffective when the available calcium is diminished.

Etiologic Factors in Sterility.—During the last four years Kotz and Parker studied completely 150 patients with the primary complaint of sterility. The etiologic factors in sterility in the female are grouped as (1) constitutional abnormalities, (2) organic pelvic pathologic conditions, (3) closed tubes and (4) endocrine abnormalities. In the male sexual incompetence is usually the etiologic factor. In every sterile couple a multiplicity of factors were operative. Absolute sterility was always due primarily to a specific organic pelvic pathologic condition. Relative sterility was usually due to many less serious factors. The secondary sterilities, as judged from their past histories, had a lowered fertility irrespective of the factors operative at the time of the examination. In the relative sterilities, pathologic changes in the pelvis, closed tubes, endocrine factors and male incompetence were the causes of the sterility, the significance of each being dependent on the severity of the condition and its association with other factors. Sterility due to a single fault was rare. Constitutional factors were not found to be the cause of sterility in any case. Endocrine factors were found to be important in the production of sterility. Blood hormone tests are valuable in determining the etiology of ovarian failure. The endometrial biopsy is the most accurate method of determining the type and degree of ovarian failure. There is no specific routine treatment for sterility. The one aim is to correct as many faults as possible. Of the 120 patients with relative sterility, thirty received no treatment. Twelve patients did not follow the treatment for the prescribed time. Seventy-eight patients with relative sterility were treated. There have been forty-one pregnancies, thirty-five of which were normal, five were miscarriages and one was ectopic. There were no instances of fetal abnormalities. Thirteen cases were considered failures and twenty-four patients are still under treatment. Of the thirty cases of absolute sterility the specific causes were as follows: The tubes had been previously removed in four, a large fibroid uterus was present in four, there was one case of bilateral chocolate cysts of the ovaries and one of sclerotic

ovaries, there were thirteen instances of closed tubes which could not be opened, pelvic inflammatory disease with closed tubes accounted for five cases and there were two cases in which male incompetence was the only cause.

Trichomonas Bartholinitis and Skeneitis.—According to Shelanski and Savitz, Trichomonas vaginalis has been demonstrated in Bartholin and Skene's glands and is a specific factor in producing bartholinitis and skeneitis. The presence of Trichomonas vaginalis in these glands is the source for reinfestation in recurrent cases of Trichomonas vaginalis vaginitis. Injection of the glands with a 1 per cent solution of silver picrate has been found useful in treating this infestation.

American Journal of Public Health, New York

29: 95-196 (Feb.) 1939. Partial Index

Duration of Immunity Conferred by Typhoid Vaccine: Results of Revaccination by Intracutaneous Injection of Typhoid Vaccine. J. F. Siler and G. C. Dunham, Washington, D. C.—p. 95.
Serologic Tests in Diagnosis of Syphilis. Elizabeth Maltaner, Albany, N. Y.—p. 104.
Studies on Trichinosis: XI. Epidemiology of Trichinella Spiralis Infestation and Measures Indicated for Control of Trichinosis. W. H. Wright, Washington, D. C.—p. 119.
Preparation of Scarlet Fever Toxin. Mary E. Evans and R. Y. Gottschall, Lansing, Mich.—p. 139.
Results of Venereal Disease Control in Canada. G. Bates, Toronto.—p. 143.
Industrial Hygiene Program in a State Health Department. C. A. Nau, Austin, Texas.—p. 151.

Archives of Ophthalmology, Chicago

21: 409-576 (March) 1939

*Common Wart as Etiologic Factor in Certain Cases of Conjunctivitis and Keratitis. A. de Rötth, Chicago.—p. 409.
*Ocular Importance of Sarcoid: Its Relation to Uveoparotid Fever. F. B. Walsh, Baltimore.—p. 421.
Spatial Disorientation with Homonymous Defects of the Visual Field. M. Kanzer and M. B. Bender, New York.—p. 439.
The Hughes Procedure for Rebuilding a Lower Lid. S. R. Gifford, Chicago.—p. 447.
Localizing Value of Incongruity in Defects in Visual Fields. D. O. Harrington, San Francisco.—p. 453.
Syndrome of Tuberous Sclerosis: Report of Case. F. L. P. Koch and M. N. Walsh, Rochester, Minn.—p. 465.
Operative Treatment of Radiation Cataract. A. B. Reese, New York.—p. 476.
Fusional Movements: Role of Peripheral Retinal Stimuli. H. M. Burian, Hanover, N. H.—p. 486.
Precocious Cataracts and Scleroderma (Rothmund's Syndrome; Werner's Syndrome): Report of Case. S. A. Agatston and S. Gartner, New York.—p. 492.
(*a*) Leiomyoma and (*b*) Hematoma of the Iris: Report of Cases. E. C. Ellett, Memphis, Tenn.—p. 497.
Fundus Oculi in Generalized Hypertension and Arteriosclerosis. R. Salus, Prague, Czechoslovakia; translated by E. Waldstein, New York.—p. 505.
Vertical Prism Imbalances in Bifocal Lenses Coexistent with Hyperphoria. S. L. Olsho, Philadelphia.—p. 515.
Diseases of the Choroid. M. Cohen, New York.—p. 522.

Common Wart in Etiology of Conjunctivitis and Keratitis.—De Rötth summarizes his experience with ocular diseases caused by common warts of the eyelids, which he first described in 1933. He has encountered seven cases of unilateral conjunctivitis, mostly of the subacute form, and three cases of keratitis of different types in connection with these warts. An additional case of bilateral conjunctivitis is reported in which warts occurred on the lids of both eyes. The virus of the wart is considered to be the etiologic factor in these cases. This view is based on the conformity of the following facts: the unilateral appearance; the involvement of the superficial layers of the eye, which points to an exogenous factor; the resistance toward the usual treatment; the presence of one or several warts on the margin of the eyelid of the diseased eye; the quick recovery after the removal of the warts, and the analogy to molluscum conjunctivitis. On the basis of these facts, the author believes that all inclusion diseases of the skin are able to attack the eye.

Ocular Importance of Sarcoid.—Walsh cites several cases which give, he believes, further clinical and histologic evidence that uveoparotid fever and sarcoid are closely related diseases. The protean ocular manifestations of uveoparotid fever and sarcoid are exemplified by the case reports. Although ocular tissues were involved in all the cited cases, many cases of sarcoid occur in which the eyes are not affected. Therefore he believes that it is no longer necessary to labor the point that uveoparotid fever and sarcoid are similar diseases and may be different manifestations of the same disease. Sarcoid and uveoparotid fever are closely allied conditions for which the cause is not known, but it will probably be proved that they are due to tuberculosis.

Arkansas Medical Society Journal, Fort Smith

35: 187-208 (March) 1939

The Female Castrate: Postoperative Care from an Endocrine Standpoint for the General Practitioner. G. R. Siegel, Clarksville.—p. 187.
Id. E. H. Hunt, Clarksville.—p. 189.
Immunology and Laboratory Diagnosis of Syphilis. M. J. Kilbury, Little Rock.—p. 191.

California and Western Medicine, San Francisco

50: 81-168 (Feb.) 1939

Experiences with Colectomy. G. D. Delprat and A. Weeks, San Francisco.—p. 87.
Miliary Lesions of the Lung, Roentgenographically Considered. R. A. Carter, Los Angeles.—p. 94.
Suicide. F. G. Lindemulder and F. E. Toomey, San Diego.—p. 98.
Auscultation of the Abdomen. J. H. Woolsey, Woodland.—p. 105.
California State Hospitals: The Problem of Overcrowding. A. J. Rosanoff, Sacramento.—p. 109.
Immunization Procedures: Report of the Committee. E. B. Shaw, San Francisco; P. Hamilton, Alhambra, and H. E. Stafford, Oakland.—p. 112.
Blind Laws of California: Some Comments. J. P. Lordan, Los Angeles.—p. 114.
Plea for Conservative Obstetrics. A. Bernstein, San Francisco.—p. 118.

Delaware State Medical Journal, Wilmington

11: 17-34 (Feb.) 1939

The National Health Program and American Medicine. M. Fishbein, Chicago.—p. 17.
Biologic Aspect of Traumatic Injuries. N. R. Washburn, Milford.—p. 28.

Florida Medical Association Journal, Jacksonville

25: 369-420 (Feb.) 1939

Diagnosis and Treatment of Amebic Dysentery. J. L. Borland, Jacksonville.—p. 381.
Disturbances of Cardiac Rhythm. S. M. Salley, Miami.—p. 387.
Adolescent Turmoil: Agitated Depression with Panic Reaction. J. V. Cohn, Hollywood.—p. 393.
Prostatic Calculi. P. D. Melvin, Miami.—p. 396.
Acute Empyema Thoracectomy. C. D. Whitaker, Marianna.—p. 398.

Georgia Medical Association Journal, Atlanta

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Crystalline Insulin. M. Sahyun, Detroit.—p. 39.
Georgia Degreed Physicians Previous to 1830. Cecelia C. Mettler, Augusta.—p. 43.
Treatment of Varicose Veins: Recent Trends. M. M. Hagood, Marietta.—p. 47.
Diagnosis and Modern Handling of Patients with Cutaneous Syphilis. H. S. Alden and J. W. Jones, Atlanta.—p. 49.
*Symptoms Occurring with Sulfanilamide Relieved by Nicotinic Acid: Preliminary Report. A. P. McGinty, G. T. Lewis and M. R. Holtzclaw, Atlanta.—p. 54.
Contraception. M. O. Corbitt, Augusta.—p. 56.
Operation for Rectal Prolapse: Report of Case. H. W. Long, Milledgeville.—p. 58.
Osteomyelitis and Compound Fractures: Treatment with Zinc Peroxide and Cod Liver Oil. P. B. Wright, Augusta.—p. 59.
Obstetrics in the Home. R. B. Crichton, Augusta.—p. 61.
Hypertrophic Pulmonary Osteo-Arthropathy: Report of Case. E. D. Hollar and E. B. Agnor, Atlanta.—p. 63.
Nissl Substances in the Nerve Cells of Teratoma. J. Krafka Jr., Augusta.—p. 64.

Sulfanilamide Reactions Relieved by Nicotinic Acid.—If the administration of nicotinic acid to patients suffering from pellagra or radiation sickness will ameliorate the symptoms and decrease or eliminate the excretion of abnormal porphyrin, McGinty and his colleagues wondered whether it would have the same desirable effect on patients ingesting sulfanilamide. They have given nicotinic acid to a number of Negro patients receiving large doses of sulfanilamide while they were confined to bed in the hospital. A control urine specimen was obtained before sulfanilamide was administered. During the period of observation, symptoms were carefully noted. On the fourth day of medication, in addition to the sulfanilamide, administration of nicotinic acid was begun in doses of 50 mg. three times a day. Throughout the period of this regimen twenty-four hour specimens of urine were collected and an oxalated specimen of blood was obtained daily. Quantitative determinations of sulfanilamide were made on the blood and urine specimens by the method of

Marshall and Litchfield. Urinary porphyrin was detected by ether extraction of an acidified sample of urine, concentration of the ether extract, acidification with 10 per cent hydrochloric acid and examination with the spectroscope. The response of the patients to the additional administration of nicotinic acid has been most gratifying. The unpleasant symptoms and the porphyrinuria were decreased. The most definite clinical change observed was a clearing of mental apathy so often present with the ingestion of sulfanilamide.

Iowa State Medical Society Journal, Des Moines

29: 47-90 (Feb.) 1939

Brain Tumors in Children. P. C. Bucy, Chicago.—p. 47.
Scientific Tests for Intoxication: Further Studies. H. W. Morgan, Mason City.—p. 53.
Latent Infections of the Mastoid. F. F. Agnew, Independence.—p. 55.
Institutional Epidemic Mastoiditis. S. G. Hands, Davenport.—p. 58.
What Do We Mean by Antiseptic Surgery? H. W. Orr, Lincoln, Neb. —p. 60.

Journal of Bacteriology, Baltimore

37: 111-236 (Feb.) 1939

Precursors to the Formation of Creatinine by Bacteria. C. H. Fish and T. D. Beckwith, Los Angeles.—p. 111.
Comparative Study of Use of Varying Concentrations of Agar in Test Medium Used to Detect Contaminants in Biologic Products. Carolyn R. Falk, Helen Bieniasz Bucca and Margaret P. Simmons, New York. —p. 121.
Double-Zone Beta Hemolytic Streptococci: Their Cultural Characteristics, Serologic Grouping, Occurrence and Pathogenic Significance. J. H. Brown, Baltimore.—p. 133.
Physiologic Youth as Important Factor in Adaptive Enzyme Formation. C. P. Hegarty, Ithaca, N. Y.—p. 145.
Method for Producing Increased Carbon Dioxide Tension in Individual Culture Tubes and Flasks. H. J. Shaughnessy, Denver.—p. 153.
Growth Factor Requirements of the Root Nodule Bacteria. P. M. West and P. W. Wilson, Madison, Wis.—p. 161.
Determination of Histamine in Bacterial Cultures. A. H. Eggerth, R. J. Littwin and Joyce V. Deutsch, Brooklyn.—p. 187.
Production of Histamine in Bacterial Cultures. A. H. Eggerth, Brooklyn. —p. 205.

Journal of Immunology, Baltimore

36: 83-192 (Feb.) 1939

Anaphylaxis in the Rhesus Monkey: I. Horse Serum as an Antigen. Lenore M. Kopeloff and N. Kopeloff, New York.—p. 83.
Id.: II. Egg White as an Antigen. Lenore M. Kopeloff and N. Kopeloff, New York.—p. 101.
Study of Meningococcus Cultures from Horses Immunized Against Meningococci. Sophia M. Cohen, Albany, N. Y.—p. 129.
Antigenic Relationships of Pneumococci to Erythrocytes and Organs of Men and Animals. A. J. Weil and E. Sherman, Pearl River, N. Y. —p. 139.
Neutralization of Vaccine Virus by Serum of Vaccine-Immune Animals. R. F. Parker, Cleveland.—p. 147.
Nonspecific Factors in Resistance: I. Capacity to Sustain Effective Circulation. A Locke, Pittsburgh.—p. 159.
Id.: II. Ability to Withstand Shock. A. Locke and E. R. Main, Pittsburgh.—p. 173.
Id.: III. Capacity for Retarding Bacterial Proliferation. A. Locke, E. R. Main and R. R. Mellon, Pittsburgh.—p. 183.

Laryngoscope, St. Louis

49: 69-134 (Feb.) 1939

Diseases of the Larynx: Material Abstracted During the Year 1938. H. B. Orton, Newark, N. J.—p. 69.
Indication for Surgery in Meningitis Secondary to Disease of the Middle Ear and of the Nasal Sinuses; Reservations in Early Surgical Intervention. H. M. Goodyear, Cincinnati.—p. 102.
Internal Jugular Vein Thrombosis Secondary to Lateral Pharyngeal Abscess: Case Report. G. B. Gilmore, New York.—p. 106.
Observation on Behavior of Leukocytes in Nasal Mucus (Preliminary Report). H. A. Schatz, Philadelphia.—p. 111.
*Ionization for Control of Severe Hypertensive Epistaxis. A. L. Beck, New Rochelle, N. Y.—p. 113.
Adamantinoma of the Superior Maxilla with Intracranial Extension. S. Androp, Baltimore.—p. 119.
Procaine Idiosyncrasy and Cervical Phlegmon as Complications Following Local Anesthesia for Tonsillectomy: Case Report. H. D. Harlowe, Virginia, Minn.—p. 123.

Ionization for Hypertensive Epistaxis.—Beck has controlled severe hypertensive epistaxis by ionization in three cases in which ligation of the external carotid was considered. The ionization, or iontophoresis, as has been employed for vasomotor rhinitis and hay fever, brought about permanent control of the epistaxis. The first was done nearly three years ago and there has been no epistaxis since in any of the patients. All three patients have a well marked arteriosclerosis. The thick fibrinous membrane of coagulative necrosis that immediately forms on the surface seems to act as a perfect blanket or cover and seals the leaking vessels. Whatever undesirable changes are produced in the mucous membrane may be considered the lesser of two evils, for control of the bleeding is the immediate necessity. The resulting submucosal fibrosis probably prevents repetitions of the bleeding. In all the cases, packing had been inserted repeatedly in an endeavor to stop the bleeding. In carrying out the procedure the packing which has been placed in the nose to control bleeding is removed a little at a time and to the enlarging nasal surface cocaine and epinephrine solution is applied topically. It is advisable to anesthetize the opposite side of the septum when only one side is involved. The ionizing pack and incorporated electrode are then introduced in the customary manner and the current is applied. A current strength of 10 milliamperes for ten minutes produces a good surface membrane. After-treatment consists of deliberate nonintervention, particularly while the pseudomembrane is present. Sedatives or narcotics are usually necessary from the first twelve to twenty-four hours. After the membrane has come away an attempt at preventing the formation of adhesions which tend to form at places at which the space is very narrow is justified. They do no particular harm.

Mental Hygiene, Albany, N. Y.

23: 1-176 (Jan.) 1939. Partial Index

Significance of Research in Mental Disorders. A. Gregg, New York. —p. 1.
General Principles of Psychotherapy. E. G. Billings, Denver.—p. 25.
The Child Reveals Himself Through Play: Method of Play Interview. J. H. Conn, Baltimore.—p. 49.
Case Work in an Authoritarian Setting. J. Slawson, New York.—p. 70.
Can the Alcoholic Become a Moderate Drinker? Anonymous.—p. 80.
Results and Problems of Group Psychotherapy in Severe Neuroses. P. Schilder, New York.—p. 87.
Experiment in Training Nurses to Help Mothers in Preventive Mental Hygiene. J. Levy, Trenton, N. J.—p. 99.

New England Journal of Medicine, Boston

220: 221-268 (Feb. 9) 1939

An Episode in Massachusetts in 1818 Related to the Teaching of Anatomy. F. C. Waite, Cleveland.—p. 221.
*Cerebrospinal Fluid in Optic Neuritis, "Toxic Amblyopia" and Tumors Producing Central Scotomas. A. L. Watkins, Boston.—p. 227.
Regional Enteritis: Study of Five Cases. W. A. R. Chapin, Springfield, Mass.—p. 232.
Polyposis of the Small Intestine: Report of Five Cases. E. A. Shaw, Providence, R. I.—p. 236.
Gynecology. J. V. Meigs, Boston.—p. 242.

Cerebrospinal Fluid and Vision.—From his examination of 120 patients who came to the hospital because of failing vision, the diagnosis in forty of whom was acute retrobulbar neuritis, Watkins concludes that the cerebrospinal fluid in acute cases is normal except in those caused by multiple sclerosis. In these, which form from 25 to 50 per cent of the group, the colloidal gold curve may show a strong first zone reaction. In thirty-nine cases of chronic retrobulbar optic neuritis the onset of the visual disturbance was slow and gradually progressive over a period of months rather than weeks. The average age (39) was somewhat higher than in the acute cases (33), with 62 per cent of patients more than 40 years of age. Both eyes were affected slightly more frequently (54 per cent) than one alone and the eyeballs were tender and painful in only 22 per cent. Pallor of the optic disks was usually observed (72 per cent), but they were hyperemic in 21 and normal in 7 per cent. The characteristic field was a central scotoma, and the visual impairment showed progression to nearly complete blindness. Lumbar punctures revealed no abnormality in initial pressure, cellular content, total protein or colloidal gold curve. The etiology was unknown in 85 per cent of the cases and proved to be multiple sclerosis in 15 per cent. The average age in the thirty cases diagnosed was 52. The course was one of slow progressive loss of vision in the two eyes, although not necessarily equally. The eyeballs were in no case tender and the optic disks were either normal or slightly pale. The diagnosis was made from visual field examinations, the characteristic field being a cecocentral scotoma, although in some cases it was made from a history of overindulgence in alcohol and tobacco and the presence of a central scotoma similar to retrobulbar neuritis, and 40 per cent of these patients improved after reducing or stopping the use of tobacco or alcohol. Cere-

brospinal fluid examinations showed initial pressures which were usually normal. The total protein averaged 33 mg. per hundred cubic centimeters, being distinctly abnormal in only two cases. The colloidal gold curve was negative in all cases. Five patients showed a diminution of vision, papillitis and a constricted field of vision. These five patients were between 4 and 44 years of age. There was tenderness of the eyeball in only one case. There was slight choking of the optic disks and peripheral constriction of the visual fields and some had only unilateral involvement. Lumbar punctures showed normal initial pressures. All cases were of fairly rapid onset, and normal vision was regained. A pansinusitis occurred coincidentally in two cases and hay fever in another. One patient had a definite lead intoxication which seemed of etiologic significance. Of six other patients with optic neuritis, three had transverse spinal cord lesions and retrobulbar neuritis which was thought to be on an unclassified infectious basis but fitted into the clinical syndrome of optical neuromyelitis. The other three had blindness with central scotomas and multiple peripheral neuritis. A necropsy in one of these cases revealed extensive periarteritis. In the three cases of optical neuromyelitis and the two of multiple peripheral neuritis with optic neuritis there was definite abnormality of the cerebrospinal fluid. In the case of periarteritis the fluid was normal. In an additional twenty-two tumor cases and failing vision the author found that the cerebrospinal fluid pressure, cellular content and colloidal gold curve may all frequently be within normal limits but that the total protein is usually increased from two to five times the normal when there is present a central scotoma from the pressure of tumors or aneurysms on the optic nerves or chiasm.

Public Health Reports, Washington, D. C.

54: 205-244 (Feb. 10) 1939

Epidemiologic Study of Poliomyelitis in the District of Columbia. C. C. Dauer.—p. 205.
*Mottled Enamel in South Dakota. H. T. Dean, E. Elvove and R. F. Poston.—p. 212.
Effect of Artificial Temperatures on Stability of Neoarsphenamine. T. F. Probey and W. T. Harrison.—p. 228.

Mottled Enamel in South Dakota.—Dean and his associates state that there are forty-one communities in South Dakota, divided among twenty counties, in which endemic mottled enamel has been demonstrated by survey. In addition there are thirty other places in which mottled enamel is probably endemic. The endemicity is seemingly limited to the smaller communities and rural districts in which the inhabitants obtain their domestic water from the Dakota sandstone, that is, users of artesian water.

Radiology, Syracuse, N. Y.

32: 131-260 (Feb.) 1939

Application of Sialography in Non-Neoplastic Diseases of the Parotid Gland. J. V. Blady and A. F. Hocker, New York.—p. 131.
Roentgen Therapy by the Method of Chaoul. E. P. Pendergrass, P. J. Hodes and C. J. Garrahan, Philadelphia.—p. 142.
Cholecystography: Single Dose versus Double Dose Methods of Administering the Dye. M. Feldman, Baltimore.—p. 155.
Influence of Ultraviolet Rays on Body Weight. F. Ellinger, Copenhagen, Denmark.—p. 157.
Treatment of Carcinoma of Breast: Technic, Complications and Results. Elizabeth Newcomer, Denver.—p. 161.
Paraplegia from Erosion of Vertebral Column by Large Thoracic Aneurysm. M. J. Hubeny and P. J. Delano, Chicago.—p. 171.
The Pulmonary Artery: Roentgenographic and Roentgen Kymographic Study. S. Brown, J. E. McCarthy and A. Fine, Cincinnati.—p. 175.
Cross Sectional Radiography of Heart. G. Danelius, Chicago.—p. 190.
*Treatment of Erysipelas with Ultraviolet Energy. M. E. Knapp, Minneapolis.—p. 195.
Diagnostic Pneumothorax. S. Schnur, New York.—p. 198.
Effect of Cable Length on Radiation Output of Shock-Proof X-Ray Tubes. T. H. Rogers, Springdale, Conn.—p. 202.
Use of Roentgen Therapy in Carotid Sinus Syndrome. C. A. Stevenson, Temple, Texas.—p. 209.
Median Rhomboid Glossitis. J. F. Sammet, Chicago.—p. 215.
Value of Assessing and Prescribing Dosage in Radiation Therapy in Simple Terms. R. Paterson, Manchester, England.—p. 221.

Treatment of Erysipelas with Ultraviolet Energy.—Knapp combines the reports of Titus and Jenkins (who treated eighty-one and fifty cases, respectively) with that of his own (510 cases) on the efficacy of ultraviolet radiation in the treatment of erysipelas. Experience shows that the dosage should be heavy so that the original dose suggested by Ude (twice the erythema dose) has now been increased to from ten to twenty times the erythema dose. A border of 3 or 4 inches of normal skin around the involved area is included in the treatment. If multiple exposures are necessary, the edges are allowed to overlap. If the eyelids are not involved the eyes are covered with black paper or cotton to prevent an ultraviolet conjunctivitis. If the eyelids are involved the patient is instructed to keep his eyes closed during the treatment. Moist packs are prohibited for from twelve to twenty-four hours after the treatment, as they interfere with the proper development of the erythema. Usually no packs are necessary. If the skin feels stiff after the erythema develops, a little petrolatum may be applied to relieve it. If the lesion spreads, the same or an even heavier dose of ultraviolet is given the next day. Most of the patients remark about the relief of pain, which often occurs while the lamp is still being applied. Later there is some increased edema of both the area of erysipelas and the surrounding normal skin. This subsides in from twenty-four to forty-eight hours, and if no further spread or complications occur the temperature drops rapidly to normal, the treated area wrinkles and then desquamates, and in five or six days the entire area is replaced with new skin. In the majority of cases only one treatment is necessary. A comparison of the three series shows that the average number of treatments per case (from 1.3 to 2.06) and the average number of days (from 3.13 to 4.58) from treatment to normal temperature are nearly the same in the three series. The percentage of deaths (7.4) is much greater in the Minneapolis series than in the others (2.4 and 2). It is probable that this can be explained by the fact that Titus and Jenkins' patients were largely private cases, while the author's series was composed entirely of charity patients, many of whom waited until the last moment before allowing themselves to be taken to a hospital. Eighteen of the deaths occurred in cases in which some factor other than erysipelas was the primary cause of death. The greatest value of ultraviolet is shown in the treatment of erysipelas in infants. Erysipelas has always been feared as an extremely fatal disease in small children. Of fifteen children less than 1 year of age treated with ultraviolet radiation, only two died. To this may be added three private cases without a death.

Rhode Island Medical Journal, Providence

22: 19-38 (Feb.) 1939

Hepatobiliary Syndromes. E. A. Field, Providence.—p. 19.
The Art of General Anesthesia. P. A. Kees, Springfield, Mass.—p. 27.

Southwestern Medicine, El Paso, Texas

23: 35-66 (Feb.) 1939

Treatment of Nephroptosis. C. H. Arnold and L. V. Gibson, Lincoln, Neb.—p. 35.
Perinephric Abscess with Ureteral Obstruction. K. D. Lynch and R. F. Thompson, El Paso, Texas.—p. 38.
Lateral Gastroduodenostomy in Surgical Treatment of Duodenal Ulcer. V. C. Hunt, Los Angeles.—p. 39.
Surgical Management of Acute Appendicitis. J. R. Phillips, Houston, Texas, and L. F. Knoepp, Beaumont, Texas.—p. 44.
Medical Care for Low Income Groups. J. R. Scott, Albuquerque, N. M.—p. 46.
*Presence of Coccidioidal Infection in Phoenix. E. W. Phillips, Phoenix, Ariz.—p. 48.

Coccidioidal Infection in Phoenix.—Phillips has encountered eight typical delayed positive reactions to coccidioidin in the district of Phoenix, Ariz. Two of these positively reacting patients have spent a summer in California within the last two or three years; another lived there for eight years and probably became infected there. Of the eight positive reactors, seven are known to be sensitized to foods, pollens or both. The remaining patient has symptoms and physical signs (including persistent eosinophilia) strongly suggesting the presence of atopic disease but the offending organism has not been found. In three of the allergic patients, prompt relief of symptoms followed the positive reaction. Another, with an excessive local reaction, suffered for three days from malaise, generalized aching and nausea, but no fever. This evidence justifies the suspicion that coccidioidomycosis is present in Phoenix, apparently in a chronic form not previously described. Unless the coccidioidin test is not specific, this organism is a factor in certain cases of asthma and chronic bronchial infection.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Medical Journal, London

1: 257-312 (Feb. 11) 1939

Virus Diseases. G. M. Findlay.—p. 257.
Intra-Uterine Aspiration. B. Lorincz and A. Davis.—p. 261.
Direct Inguinal Hernia. W. G. Gill.—p. 263.
*Transmission of Syphilis by Blood Transfusion. J. A. W. McCluskie.—p. 264.
Suggested Skin Protective. P. B. Mumford.—p. 266.
Clinical and Social Survey of Male Linen Weavers. J. Deeny and H. S. Booker.—p. 267.

Transmission of Syphilis by Blood Transfusion.—McCluskie reports a case of syphilis transmitted by a blood transfusion. The disease did not show itself until two months after transfusion, unless a few reddish macules on the arm were early secondary manifestations—syphilis d'emblée. In both the donor and the recipient the disease responded to relatively small doses of arsenic and bismuth preparations. The Wassermann reactions of the donor and the patient were both tested at the time of the transfusion and were negative, and neither presented any sign of the disease. The donor had jaundice shortly after giving the blood, and the recipient two weeks after transfusion. Two other authentic cases are reviewed from which it is clear that syphilis can be transmitted from donors who are clinically and serologically negative. This fact should be explained to prospective donors, so that, by careful questioning, one may be able to rule out those who have been exposed to infection during the preceding two months. It has been suggested that the greatest safeguard against such accidental transmission of syphilis would be to employ cadaver blood obtained according to the technic of Yudin (1936). Apart from the natural prejudice which the general public in England might have to the practice, it would be possible only when conducted on a large scale with considerable laboratory facilities. It may in the future be practicable to add some innocuous antiseptic—for example, acriflavine—to the blood for a sufficient period before transfusion to ensure sterility. The author has added a small amount of neoarsphenamine (0.45 Gm.) to the transfused blood in emergency cases in which there has been doubt with no untoward effect. These measures were purely empirical, and he has no proof of their efficacy. In addition to the risk to the patient there is the possibility of an action for damages against the doctor who transmits syphilis by transfusion.

Lancet, London

1: 249-308 (Feb. 4) 1939

X-Ray Kymography of the Heart. F. G. Wood.—p. 249.
*Treatment of Pneumonia with Sulfapyridine. T. F. Anderson, with bacteriologic observations by R. M. Dowdeswell.—p. 252.
Chronic Constrictive Pericarditis. J. B. Hunter and T. East.—p. 255.
Ossification of the Vertebral Body. T. J. B. A. MacGowan.—p. 258.
Neoplastic Cysts Communicating with the Ventricular System: Report of Case. W. McKissock.—p. 259.
Metabolic Eczema in Idiopathic Steatorrhea. I. D. Riley.—p. 262.
Osteomalacia of the Spine Associated with Chronic Diarrhea. J. M. Holmes.—p. 264.
*Chemotherapy in Plague Infection. H. Schütze.—p. 266.

Sulfapyridine in Pneumonia.—Anderson and Dowdeswell compare the results in fifty cases of pneumonia treated with sulfapyridine with fifty cases in which nonspecific treatment was given. There were eight deaths in the latter series as compared to one in the series treated with sulfapyridine. The course of the disease was considerably modified by sulfapyridine and the pyrexial period was reduced. The drug appeared to be active against all types of pneumococci.

Chemotherapy in Plague Infection.—Schütze tested the efficacy of a benzylsulfanilamide derivative, sulfapyridine and a diaminodiphenylsulfone glucoside against infection (experimental) with Bacillus pestis in mice and rats. All three compounds produced a definite therapeutic effect; sulfapyridine was more potent than either in both rats and mice. The benzylsulfanilamide derivative protected rats but not mice and the diaminodiphenylsulfone glucoside protected mice but not rats. Antiplague serum injected subcutaneously at the same time as the test dose of Bacillus pestis produced results comparable with those of sulfapyridine. Future experiments may show whether a combination of the two treatments is superior to either alone.

Medical Journal of Australia, Sydney

1: 131-172 (Jan. 28) 1939

Eye, Ear and Fingertip. W. A. Osborne.—p. 131.
Alcoholism and Its Treatment. J. Bostock.—p. 136.
Notes on "Cardiazol" Therapy in Schizophrenia. C. R. D. Brothers and Isabel Williams.—p. 138.
The Stiff Joint. E. B. M. Vance.—p. 139.
Studies in the Urinary Excretion of Vitamin C in Certain Diseases of Children. S. Williams and Margaret Green.—p. 145.
Role of the Bandicoot in the Epidemiology of Q Fever: Preliminary Study. E. H. Derrick, D. J. W. Smith, H. E. Brown and Mavis Freeman.—p. 150.

Quarterly Journal of Medicine, Oxford

8: 1-78 (Jan.) 1939

Spontaneous Pneumothorax. K. M. A. Perry.—p. 1.
*Insulin Resistance and Diagnosis of Thyroid Disease. W. J. Griffiths.—p. 23.
Further Studies of Plasma Lipids in Toxic Goiter: Evidence Suggesting a Bimodal Distribution. E. M. Boyd and W. F. Connell.—p. 41.
Plasma Lipids in Anxiety States and Their Comparison with Lipopenia of Hyperthyroidism. E. M. Boyd, W. F. Connell and A. M. Doyle.—p. 47.
Obesity, Hypogenitalism, Mental Retardation, Polydactyly and Retinal Pigmentation: The Laurence-Moon-Biedl Syndrome. A. Sorsby, H. Avery and E. A. Cockayne.—p. 51.
Substitution Therapy in Hypopituitarism. A. W. Spence and L. J. Witts.—p. 69.

Insulin Resistance and Diagnosis of Thyroid Disease.—Griffiths describes an abnormality of carbohydrate metabolism in thyroid disease which can be detected even in the absence of any significant increase in the basal metabolic rate. This abnormality takes the form of a diminution in the sensitivity of the muscles to insulin. Thus, whereas in the normal subject the rate at which the muscles withdraw sugar from the blood is greatly increased by insulin, this effect is either diminished or abolished in thyrotoxic and in many nonthyrotoxic cases of goiter. The author bases his remarks on the data obtained from a study of thirty-five cases of thyroid disease. In toxic goiter the peripheral insulin response to the insulin-dextrose test is either poor or negligible, denoting an impairment of the power of the muscles to utilize carbohydrate; this is referred to as peripheral insulin resistance. A similar condition of resistance is found in many cases of nontoxic goiter in which, although the basal metabolic rate is not raised, other signs suggestive of thyroid disturbance, such as intermittent tachycardia, increased fatigability, nervousness and the like, are present. In some cases of goiter, both toxic and nontoxic, central resistance to insulin is present. This condition may be described as an enfeeblement of the normal action of insulin in promoting storage of sugar in the liver. With few exceptions, in both types of cases peripheral insulin resistance is considerably diminished by subtotal thyroidectomy. Central resistance is also in general abolished by thyroidectomy, but again it is occasionally unaffected. The possible relationship between peripheral insulin resistance and myasthenia is stressed. In this connection the creatinuria which is a common feature of thyroid disorders is of particular interest, more especially in view of the association of peripheral insulin resistance, creatinuria and myasthenia in other diseases such as diabetes, acromegaly and dermatomyositis. In view of the frequency with which the author has encountered peripheral insulin resistance in cases in which the nonthyrotoxic or psychoneurotic element of goiter enters, he suggests that estimation of this factor might prove to be a valuable method of detecting possible aberrations of thyroid function in patients who, while not thyrotoxic in the sense that they have a raised metabolism, nevertheless complain of weakness, increased fatigability and symptoms of a cardiac or nervous character.

South African Medical Journal, Cape Town

13: 37-78 (Jan. 28) 1939

National Health Insurance and an Improved Rural Medical Service. H. Gluckman.—p. 39.
State Medical Insurance in British Columbia. W. Wilson.—p. 41.
A Few Personal Thoughts on General Practice—Past and Present. C. E. L. Burman.—p. 44.
Laryngeal Diphtheria. P. Bayer.—p. 47.
Medical Practice Among Natives. D. D. Dunn.—p. 51.
The Future of Tuberculosis Control: The Papworth Demonstration. N. Mann.—p. 53.
The Transkeian Heart: Tuberculous Pericarditis. J. C. Downes.—p. 55.

Journal de Chirurgie, Paris

53: 305-448 (March) 1939

Tumors of Internal Theca of Ovary. P. Moulonguet and J. Varangot.—p. 305.
Abdominoperineal Amputation with Hysterocolpectomy in Cancer of Rectum. J. Charrier and Barraya.—p. 322.
Connections Between Staphylococcic Infections and Avitaminoses. J. Patel.—p. 344.
Technic of Ablation of Aorticorenal Ganglion. R. Fontaine and F. Bilger.—p. 352.
*Quincke's Edema of Viscera Simulating Acute Appendicitis. H. Redon.—p. 360.

Quincke's Edema Simulating Acute Appendicitis.—Redon reports the clinical history of a man who had severe pains on the right side of the abdomen. His physician observed that the abdomen was supple; moreover, the patient did not vomit; the temperature was 37.5 C. (99.5 F.) and the pulse was 80. The physician recommended the application of ice and decided to wait twelve hours before making a decision. When Redon saw the patient, the general symptoms were still the same. He rejected the diagnosis of appendicitis and in view of the paroxysmic character of the pains he considered the possibility of an acute invagination and decided to resort to a surgical intervention. On opening the peritoneum he was struck by the extraordinary appearance of the ileocecal region. There was a sparkling edematous mass and Quincke's edema was thought of, the more so since there were no other abnormalities: the appendix in particular presented no sign of inflammation, and the ileum and cecum were likewise normal. However, the appendix was removed and the microscopic examination verified that it was free from anatomic abnormalities. The postoperative recovery was uneventful. The patient was discharged from the clinic on the tenth day. Later investigations confirmed the author's first impression. The patient stated that he was subject to frequent urticarial crises, especially after ingestion of fish, and that before the attack in question he had eaten fish and mussels. Thus the case seems to represent a true attack of Quincke's edema or of "visceral urticaria," as Paul Chevallier proposes to designate the condition. The author regards this case as noteworthy because it proves the existence of a visceral form of Quincke's edema, which, although assumed by some, had not been proved as yet. For surgeons, this case explains certain obscure abdominal symptoms and thus diagnostic errors may be avoided.

Presse Médicale, Paris

47: 281-304 (Feb. 22) 1939

*Gonadotropic Secretion and Functional Placental Activity: Hormonal Characteristics of Insufficiency, of Normal Condition and of Hyperactivity of Placental Gland. A. Brindeau, H. Hinglais and M. Hinglais.—p. 281.
Question of Parakeratoses, Dermatoses of Sensitization: Is Psoriasis an Ultravirus Disease? A. Desaux and H. Prétet.—p. 283.

Gonadotropic Secretion.—Brindeau and his co-workers report studies on the quantitative estimation of the gonadotropic secretion. They used successively two methods of titration: first they employed male mice and later rabbits. The latter method is at the same time convenient, rapid and precise and it constitutes in practice a veritable functional test, a method of measurement of the activity of the placental gland. The activity of the placental gland can be insufficient, normal or exaggerated. The authors take up first the secretion of the gonadotropic hormone in nonpregnant women and demonstrate that in healthy, sexually mature women traces of the gonadotropic hormone are found. However, in certain pathologic conditions, such as amenorrhea, cancer of the cervix uteri and lutein cysts or after the menopause, considerable quantities of gonadotropic hormone may be found. In some of these cases 100 or more rabbit units may be detected per liter of urine. Quantities of about 150 units, the authors regard as uncertain in their diagnostic significance, because they overlap into the zone of those values that indicate a dead ovum. In the table, in which the authors list the quantitative values of gonadotropic secretion during gravidic states, they first mention the amounts that indicate placental insufficiency. In cases of placental insufficiency with certain death of the ovum, up to 500 rabbit units of gonadotropic hormone may be detected, whereas values between 500 and 2,000 units indicate weak placental action but not necessarily death of the ovum. In the presence of normal placental activity between 2,000 and 25,000 units is detected. Values that exceed 25,000 units are indicative of placental hyperactivity. In placental hyperactivity the authors differentiate two groups: 1. Values between 25,000 and 60,000 units are observed in the toxicoses of pregnancy. In hyperemesis gravidarum, for instance, values between 30,000 and 50,000 units have been detected. 2. Values of 60,000 units and higher indicate the presence of hydatidiform mole. The authors show further that gonadotropic titration is an important aid in the early diagnosis of malignant chorionepithelioma. After the evacuation of the hydatidiform mole, the gonadotropic hormone content of the blood should be determined at intervals of about ten days. If the values fall to zero, the woman may be regarded as out of danger. However, if the titer remounts again after temporary decrease, malignant degeneration is certain and operation is necessary.

Arquivo de Patologia, Lisbon

10: 125-380 (Dec.) 1938. Partial Index

*Cancer of Lung. Nunes d'Almeida.—p. 221.
Roentgen Therapy in Fibroma of Uterus. Irene Nunes.—p. 351.

Cancer of Lung.—Nunes d'Almeida reports sixteen cases of cancer of the lung. The greatest incidence of the disease was between the ages of 50 and 60 years. All patients but one were men. In the majority the disease developed in the right lung. The author found that the symptoms of cancer of the lung are not precise. Early in the development of the disease the patients are in apparently good health. However, they suffer from repeated hemoptysis, frequently from acute pain in the thorax and shoulder of the side in which cancer developed or from dyspnea. All patients report a progressive thinness and cough during which solid material is eliminated. Axillary and cervical metastases may develop in some cases in the absence of symptoms of diseases of the respiratory tract. The diagnosis is made from microscopic study of the material eliminated while coughing or from material taken by biopsy from grown ganglions and confirmed by x-ray examination of the thorax followed by bronchoscopy in the frontal and lateral postures. Pulmonary cancer may be of bronchial or parenchymal (including lobar) type. Bronchial cancer is the most common and acute form in the group. It develops in the wall of the main or large bronchi, partially (or completely) occludes the lumen of the bronchi, rapidly involves the hilar mediastinal and paravertebral ganglions and the lung parenchyma, and gives early metastases. It shows in the roentgenograms by opacities of the atelectatic lobe or lobes of the lung, and during bronchography by the stopping of the progress of the opaque substance at the occluded zones. Sometimes the tumor is not visible, but the stricture of the visible bronchi indicates the presence of cancer in distant bronchi. The parenchymal forms may be either primary, which are benign, or secondary to bronchial cancer, which are acute. Pure parenchymal forms show in the roentgenograms as isolated tumors at either one or various lobes. An early differential diagnosis between bronchial and parenchymal cancer is important because of the fact that parenchymal forms, especially lobar, are amenable to cure by an early operation (lobectomy or pneumonectomy). The earlier the operation and the better the preoperative and postoperative care of the patients, the better the results. Radium locally applied and roentgen treatments are palliative. They are indicated in early bronchial cancer and in advanced parenchymal cancer.

Brasil-Medico, Rio de Janeiro

53: 89-237 (Jan.) 1939. Partial Index

*Complement Fixation in Leishmanioses. A. Marques da Cunha and E. Dias.—p. 89.
Conservative Surgery in Calculous Cholecystitis. P. Cezar de Andrade.—p. 95.
Cerebral Symptoms in Course of Acute Rheumatic Fever. A. Sampaio Tavares.—p. 97.

Complement Fixation Reaction in Leishmanioses.—Marques da Cunha and Dias state that the complement fixation reaction is not resorted to for the diagnosis of leishmaniosis because there is no proper antigen available for the test. The authors prepared a stable alcoholic antigen from Leishmania cultures. They give in detail the technic for the preparation

of the antigen. The authors found that the antigen which they prepared induces (1) strongly positive complement fixation in the blood serum of rabbits which are hyperimmunized with various species of leishmania, (2) positive complement fixation in the blood serum of patients who are suffering from American, visceral and tegumentary leishmaniosis and in animals with experimental leishmaniosis and (3) moderate positive results in the blood serum of patients who have Chagas' disease. A differentiation of the various species of Leishmania which cause the various forms of clinical or experimental leishmaniosis cannot be made by the results of the test.

Beiträge zur klinischen Chirurgie, Berlin

169: 1-176 (Jan. 31) 1939. Partial Index

Lockjaw and Gibbus. R. Löhr.—p. 1.
Diagnosis of Strain as Cause of Fracture of Spinous Process of Vertebra. P. Rostock.—p. 15.
Permanent Result of Two Stage Operation on Biliary Passages. F. Bernhard.—p. 25.
Results with Nicolas Operation for Habitual Dislocation of Shoulder. E. Kreuter.—p. 32.
*Ureteronephrectomy for Renal Tuberculosis. C. van Gelderen.—p. 52.
*Results from Use of Filtered Ultraviolet Rays in Laparotomies. T. Marti, —p. 84.

Ureteronephrectomy for Renal Tuberculosis.—According to van Gelderen there were thirty-nine cases of renal tuberculosis in which ureteronephrectomy was performed at the surgical clinic of the University of Amsterdam between 1919 and 1938. The more extensive operation did not increase either the immediate operative mortality or the mortality of the later course. The ureteral incision healed in almost every instance by primary intention and remained healed. The renal incision presented about the same incidence of wound complications as is seen after a plain nephrectomy. Apparently these complications are not due to the ureter left behind. The removal of the ureter reduces the average duration of wound complications. Empyema of the ureter, cold abscesses and postoperative colics are absent. Even the unfavorable cases do well after ureteronephrectomy. There were three fatalities in this series. The operation presented no disadvantages. The anticipated advantages, however, did not justify themselves completely. The rapid and permanent recovery of the bladder, however, is apparently due to the removal of the ureter. The author recommends this combined operation in all except the early cases of renal tuberculosis.

Filtered Ultraviolet Rays in Laparotomies.—According to Marti, clinical observations on the effect of irradiation with ultraviolet rays in laparotomized patients established the effectiveness of the method in reducing postoperative pain. The therapy, however, was not successful in every case and recourse to analgesics was necessary. Ultraviolet irradiation during the operation had a definite effect in increasing peristalsis during the postoperative period. In various forms of abdominal operations, particularly after an operation for incarcerated hernia, the treatment reduced in some degree the mortality. In cases of diffuse suppurative peritonitis, the results were far from those claimed by Havlicek, who had obtained a 100 per cent cure. The incidence of postoperative thrombosis, embolism and pulmonary complications was reduced. This was particularly striking as far as the occurrence of pulmonary embolism after operation for incarcerated hernia was concerned. The results, however, are far from those claimed by Havlicek. The author feels that the employment of the laparophos lamp in abdominal operations is a valuable therapeutic agent, even though somewhat overrated by its originator.

Medizinische Welt, Berlin

13: 177-212 (Feb. 11) 1939. Partial Index

Occupational Diseases of Lungs. R. Schoen.—p. 177.
Acute Appearance of Multiple Small Tuberculous Cavities. E. Saupe.—p. 181.
*Genesis and Therapy of Hyperemesis Gravidarum. G. Guhr.—p. 182.
Organization of Blood Donors and Blood Transfusion. K. A. Seggel and H. Reiher.—p. 185.
Quinine in Influenza. E. Pawlowski.—p. 190.

Genesis and Therapy of Hyperemesis Gravidarum.—Regarding the genesis of hyperemesis gravidarum, Guhr says that the theories about it have undergone many changes but that recent advances in the research on hormones and vitamins have introduced new points of view regarding the pathologic-physiologic processes that are involved in this disorder, and the therapeutic measures that have been based on these new theories have produced considerable success. In order to explain the treatment with vitamin B_1 and a liver extract which is being used successfully at the author's clinic, he reviews the theoretical foundations that correspond to the present insight into the complicated metabolic processes, giving special attention to the hepatic impairment. Following this discussion he reports the results obtained in twelve cases of hyperemesis gravidarum which were treated with vitamin B_1 and a liver preparation. The patients are given intragluteal injections of ampules of liver extract (2.2 cc.) which contain 500 international units of vitamin B_1. At first one ampule of the preparation is injected daily and as soon as improvement is noticeable the injections are given every second day. The total number of injections varies in different cases, but from six to ten are usually sufficient. In especially severe cases more than one injection may be given daily. The diet should be rich in carbohydrates. Discussing the mode of action of the combined administration of vitamin B_1 and liver, the author suggests that it acts as a protective liver therapy, because investigations indicate that a primary hepatic disorder and the involved disturbance in the carbohydrate metabolism are decisive factors in the genesis of hyperemesis gravidarum. Dysfunction of the anterior lobe of the hypophysis is the eliciting cause. Deficiency in vitamin B_1 is not regarded as a causal factor of hyperemesis gravidarum; however, as an adjuvant vitamin B_1 supports the action of the liver extract in that it not only favors an increase in glycogen but also normalizes the disordered carbohydrate metabolism.

Strahlentherapie, Berlin

64: 1-200 (Jan. 25) 1939. Partial Index

Investigations on Dosimetry of Ultraviolet Rays. U. Henschke and R. Schulze.—p. 14.
Results of Irradiation in Sarcomas: Observations in Zurich from 1919 to 1936. O. Walther.—p. 59.
Preventing Secondary Effects on Urinary Bladder in Ray Therapy of Cancer of Uterus. T. C. Neeff and F. Hoff.—p. 113.
Rectal Complications in Patients with Uterine Cancer. F. Gál.—p. 125.
*New Method of Irradiation by Means of Moving Radium Preparations. K. Mayer.—p. 134.
Measurements in Roentgens on Telecurie Therapy Apparatus. A. Gunsett and G. F. Gardini.—p. 149.
Biophysical Foundations of Weak Radium Therapy. B. Rajewsky.—p. 158.

Irradiation by Means of Moving Radium Preparations.—Mayer points out that irradiation by means of moving x-ray tubes was suggested as early as 1913 by H. Meyer. In spite of the great importance of this technic, it was neglected until in recent years it was revived in the form of rotation therapy by Dessauer, René du Mesnil de Rochemont and others (see also *Strahlentherapie* **60**:645, 648, abstracted in THE JOURNAL April 2, 1938, p. 1152). In the present report the author describes his own efforts in adapting the moving principle to radium therapy, pointing out that he experimented with moving radium preparations for the first time in 1926 (report in "Nowiny Lekarskie," page 428) and that in 1933 he discussed the therapeutic application of moving radium preparations. He shows that the irradiation with moving radium involves less technical difficulties than does the employment of the moving principle in roentgen therapy. The small and free radium containers can be coupled easily with a driving mechanism consisting of a clockwork or of an electrical device. It is possible to employ various types of movements. The radium container may be moved in straight lines back and forth, or a rotatory or vibratory method may be employed. The author evaluates the mode of action and the distribution of the radium rays, if the moving technic is employed, pointing out that greater uniformity of ray perfusion of the tissues reduces the danger of relapse in case of tumors. Discussing the devices of application, he says that account must be taken of the available radium supply. This applies especially to the radium containers, not so much to the driving mechanism. The author shows in illustrations that the same motor can be used to produce straight-lined back and forth movements, oscillating arc movements or rotating movements. Regarding the determination of the dosage in the treatment with moving radium preparations, the author refers to studies by René du Mesnil de Rochemont on rotatory roentgen therapy.

Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam

83: 377-476 (Jan. 28) 1939. Partial Index

Surgical Treatment of Pulmonary Tuberculosis. W. Bronkhorst.—p. 378.
*Investigations on Blood Circulation in Drumstick Fingers. W. H. D. De Haas.—p. 384.
Diagnosis and Treatment of Chronic Gonorrhea of Cervix. L. A. Falke.—p. 390.
Measuring of Light Projected Through Coagulating Blood, a Method to Determine Time and Duration of Coagulation. H. Festen.—p. 396.

Circulation in Drumstick Fingers.—De Haas says that many reports about the microscopy of drumstick fingers maintain that there is a strong overfilling of the capillaries. This is of practical significance in that it forms the anatomic basis for the widely accepted opinion that the pathogenesis of drumstick fingers is connected with stasis and cyanosis. However, there are also some investigators who found the capillaries normal or contracted. The author's own investigations aimed at determining possible differences in the brachial circulation of two groups of ten patients with bronchiectasis, in one of which drumstick fingers were present and in the other absent. The studies consisted in capillaroscopy and in the determination of the venous pressure and of the cutaneous temperature. The results of these investigations are recorded in tables. It was found that in drumstick fingers the smallest vessels are not dilated but contracted. The author thinks that this explains some of the peculiarities of drumstick fingers, for instance the localization of this symptom on the extremities that are most exposed to cold, that is, on the fingers rather than on the toes; moreover, the greater incidence in men than in women (in women the capillaries are dilated during menstruation). Regarding the cause of this vasoconstriction the author says that a detailed discussion is impossible in the scope of this article, but he suggests that the vasoconstrictive substances, of which it is known that the organism can produce them, are destroyed in the lungs. In some diseases of the lungs and in congenital heart diseases (among others in bronchiectasis) this filtrating action of the lung fails. The author thinks that it is the impairment, exclusion or overflowing of the pulmonary filter which is of decisive importance for the development of drumstick fingers.

83: 477-616 (Feb. 4) 1939. Partial Index

Treatment of So-Called Neuroses in Children. H. C. Rümke.—p. 483.
*Treatment of Uterine Prolapse in Old Women. F. C. van Tongeren.—p. 493.
Extraction of Cataract in Small Hospitals. G. Wiegersma.—p. 498.
Petrositis. P. G. Gerlings.—p. 504.

Uterine Prolapse in Old Women.—Van Tongeren shows that old women who have a uterine prolapse can often be relieved to a considerable extent by a pessary. Even in case of total prolapse the pessary may be helpful. However, if the treatment with the pessary fails, the operation according to Neugebauer-Le Fort is advisable. After this operation, relapses are less likely than is the case after the operation that has recently been recommended by Kahr. Kahr's operation is even more simple than that of Neugebauer-Le Fort. Kahr too does not counteract the prolapse as such but makes a blockage on the vulva. The author employed Kahr's operation in seven cases but in three of these a relapse resulted. To be sure, all of these women had a total descensus uteri. However, the results obtained with the operation of Neugebauer-Le Fort were more favorable, for of the twenty-five women who were operated on according to this method only three had a relapse and these could now be treated with a pessary, which had not been possible previously. Thus it seems that if relapses do follow the operation of Neugebauer-Le Fort they are usually less serious than those which develop after Kahr's operation.

83: 617-726 (Feb. 11) 1939. Partial Index

Chronic Cystic Mastitis and Carcinoma. S. M. Kropveld.—p. 626.
Treatment of Coxa Vara in Adolescents. C. P. van Nes.—p. 635.
*Amyloid Deposits in Islands of Langerhans in Diabetes Mellitus. C. van Beek.—p. 646.

Amyloid in Islands of Langerhans in Diabetes.—Van Beek was induced to make histologic studies on the islands of Langerhans of patients with diabetes mellitus by the detection of amyloid deposits in the islands of Langerhans of a woman aged 54 who died in diabetic coma. Reviewing the literature he found reports on hyaline degeneration of the islands of Langerhans in diabetic patients, but he questions that these deposits really are hyaline, for reactions in various staining tests indicate that the substance is amyloid. Studies on the pancreas of fifty-nine patients with diabetes mellitus, whose ages ranged from 5 to 85 years, disclosed amyloid in twenty-five, or 42.3 per cent. In classifying the patients according to age groups it was observed that the amyloid deposits are present only in those over 50 years of age and that with increasing age the incidence of these deposits becomes greater. The author shows that the question whether the amyloid precipitate in the islands of Langerhans is a partial manifestation of a generalized amyloidosis must be answered in the negative. However, the occurrence of occasional amyloid deposits in some pancreatic islands in aged persons, in the absence of clinical signs of diabetes, makes it difficult to decide whether amyloid deposits are the cause or the result of diabetes mellitus. A parallelism with arteriosclerosis, especially of the arterioles, could be demonstrated. The factors leading to the precipitation of amyloid are still unknown.

Ugeskrift for Læger, Copenhagen

101: 115-140 (Jan. 26) 1939

Substances in Phosphate Concrements of Urinary Tract (Chemical-Roentgenographic Study). A. T. Jensen and J. E. Thygesen.—p. 115.
*Vitamin P in Hemorrhagic Nephritis. T. Gimsing.—p. 117.
Thrombopenic Purpura After Treatment with Arsphenamine. A. Stigaard.—p. 118.
Harness for Abduction Splints. O. Raagaard.—p. 119.

Vitamin P in Hemorrhagic Nephritis.—Gimsing reports seven cases of hemorrhagic nephritis treated with injections of vitamin P. In no case were there results which could not have been attained as well by rest in bed and diet; in some cases the treatment was without effect, in others it seemed to exert a transitory irritating effect on the kidneys.

101: 141-172 (Feb. 2) 1939

*Family with Hereditary Epidermolysis Bullosa. E. D. Bartels.—p. 141.
Skin Transplantation with Film Bandage. M. Olesen.—p. 144.
Eczema Due to "White Ox-Eye" (Chrysanthemum Leucanthemum). P. V. Marcussen.—p. 147.

Hereditary Epidermolysis Bullosa.—Bartels says that in this disorder bullae appear in the parts of the skin exposed to mechanical action, most often on the hands and feet, but also, though rarely in the simple form, on other parts of the body and in the mucous membrane, as in the mouth and throat, and the nails. The content of the blisters is at first clear, later often becoming hemorrhagic. In the simple form the bullae heal completely in from one to two weeks, in the dystrophic form they are followed by more or less marked atrophy of the skin. The disorder as a rule sets in early, often shortly after birth. The prognosis for cure is unfavorable. A tendency to hyperhidrosis is often present; treatment of the hyperhidrosis leads to a not inconsiderable improvement. Of fifty-five members of the family in question twenty-five (eleven men, fourteen women) are known to have epidermolysis bullosa simplex. Transmission seems to be dominant and not connected with the X-chromosome, at least not exclusively. The author believes that ephedrine may be helpful for a definite limited time. The effect of ephedrine apparently supports the theory that there is in these cases a disturbance of the blood vessels or of the nerves and blood vessels.

101: 203-230 (Feb. 16) 1939

*Case of Streptococcic Infection After Injection for Protection Against Diphtheria. T. Madsen and E. J. Henningsen.—p. 203.
Investigations on H. Rotter's Intracutaneous Test in Man for Establishment of Vitamin C. E. Poulsen and H. Lieck.—p. 206.
Tetany in Breast-Fed Child on Basis of Latent Osteitis Fibrosa in Mother: Contribution to Function of Parathyroid Gland. C. Friderichsen.—p. 209.
Blocking and Forming of Nose in Child Aged 4. E. Dujardin.—p. 211.
Electrical "Phantom Heart." G. Rendtorff.—p. 212.

Streptococcic Infection After Diphtheria Immunization.—Madsen and Henningsen review the complications (abscesses and scarlatiniform exanthem) in thirty-four of 2,418 children given injection against diphtheria in January 1938. With one exception all were vaccinated by the same physician at the same time and place. Examination of pus from the abscesses in twenty-three cases revealed a hemolytic streptococcus belonging to group A, type II, in twenty-two cases. Investigation indicated that the anatoxin was contaminated by the assisting nurse, in whose throat and nasal secretion a streptococcus, type II, was established.